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The Symposium

In 1956, coronary disease accounted for 74,790 deaths in England and Wales compared with 58,309 in 1951—an increase of around 28 per cent. These deaths from coronary disease in 1956 represented 38.6 per cent. of all deaths from diseases of the circulatory system whilst the latter was the commonest cause of death, being responsible for 37.2 per cent. of all deaths. Expressed in another way, coronary disease was responsible for 14.3 per cent. of all deaths in England and Wales in 1956, compared with cancer which was responsible for 17.8 per cent., and diseases of the respiratory system which were responsible for 11.5 per cent. This is the challenge which faces medicine today, and which provides ample justification for a symposium reviewing the salient facts.

The most interesting development in this field in recent years has been the increasing emphasis upon the role of dietary fat in the causation of coronary disease, and this aspect of the subject is fully reviewed by Professor J. F. Brock, who has himself made some outstanding contributions to the subject. His balanced review of the problem should go far towards clarifying the issue for those many clinicians who have found it difficult to correlate the many contradictory statements which have appeared on the subject. His major conclusion is that the incidence of occlusive coronary episodes in people at risk would be reduced by modifications of diet, particularly reduction in the quantity or alteration in the quality of fat. He adds, however, the important rider that 'the evidence is not yet decisive enough to justify altered general advice on food patterns and nutrition education for apparently healthy people, or for the prevention of atherosclerosis'.

Fact and Fancy

What Dr. Donald Reid in his contribution to the symposium this month (p. 184) describes as the 'New Epidemiology of the chronic non-infectious diseases' has undoubtedly an important contribution to make to a better understanding of chronic processes. The method, however, is not without its 'hazards', as Dr. Reid rightly points out, and in view of the increasing importance being attached to this epidemiological approach to the subject, it is essential that these 'hazards' should be recognized. A particularly good example quoted by both Dr. Reid (p. 188) and Professor Brock (p. 194) is that of Morris's study of the incidence of coronary thrombosis in London bus conductors and drivers. This showed a higher incidence in drivers than in conductors which was first interpreted as evidence of an adverse effect of either sedentary occupation or tension and strain. In a subsequent report, however, Morris

produced evidence that this difference might be genetic in that recruits to driving differed in their physique from conductors.

Yet another example is a recent report from Scotland, throwing doubt on the commonly held view that coronary disease is relatively uncommon in agricultural workers. According to A. C. Kennedy (*Scot. med. J.*, 1957, **2,** 420), in the three-year period, 1954-56, of the males with coronary disease admitted to the Dumfries and Galloway Royal Infirmary, agricultural workers proved the largest occupational group (approximately 24 per cent.) of cases, and this figure corresponded very closely with the percentage of agricultural workers in the area served by the hospital (26.8 per cent.). It is anomalies such as these which indicate the need for particular care in drawing definitive conclusions from epidemiological data when dealing with a condition such as coronary disease in which a multiplicity of etiological factors is probably involved.

Chronic Illness

THE problem of chronic illness is assuming increasingly large proportions as the progress of medicine extends the expectation of life and saves the lives of, but cannot yet restore health to, many children who, fifty years ago, would have died at birth or soon afterwards. According to a recent American report, it is estimated that in 1950 there were 28 million Americans who 'were suffering from disabling and non-disabling disease or impairment', and the view is expressed that 'there is no reason to think that this number has decreased'. This report, 'Prevention of Chronic Illness' (Harvard University Press. London: Oxford University Press. Price 48*s.*), is one of a series based on the work of the Commission on Chronic Illness, which, from 1949 to 1956, studied the chronic illness problem in the United States. It is the most comprehensive study of the subject yet made, and is deserving of careful study by all who are concerned with the future welfare of the community.

It provides no ready-made solution to the problem but it does justify certain conclusions. The first is that the long-term solution is to be found in prevention and that in this connexion increasing attention requires to be given to the prevention of arterial disease, chronic rheumatism, diabetes mellitus and mental disease. If these four diseases (or group of diseases) could be prevented, the problem of chronic illness would be well on the way to solution. Such prevention is dependent upon an understanding of their cause, or causes, and therefore upon a more intensive research programme along these lines. The second conclusion that can be drawn is that, in the present state of knowledge, mass surveys of the population to discover disease in its incipient stage seldom justify the expense and energy involved. The third conclusion is that an efficient standard of nutrition, housing, and sanitation pays handsome dividends in the prevention of disease. Finally, the family doctor can do more than all the most elaborate surveys to detect disease in its early stage and to ameliorate the lot of the chronically disabled. In some ways this is the outstanding challenge of the day to the general

practitioner and one which we are sure he is as capable of meeting as his predecessors met that of the fulminating infections of fifty years ago.

Congenital Deformities

From time immemorial congenitally abnormal children have excited the morbid curiosity of mankind. Inevitably, supernatural factors were given priority as causative agents and, for long, Aristotle was a voice crying in the wilderness, with his suggestion that an explanation of these deformities might be found in a study of comparative embryology. The last thirty years have witnessed a growing volume of evidence that the Aristotelian approach was the right one and that environmental factors play an important, if not the most important, part in the causation of these distressing abnormalities.

The latest report on the subject comes from Harvard where T. H. Ingalls and F. J. Curley (*New Engl. J. Med.*, 1957, **257,** 1121) have shown that pregnant mice exposed within a low-pressure chamber produced many fœtuses with malformed ribs and vertebræ and that these defects were 'clearly due to hypoxia brought about by maternal exposures to reduced oxygen tensions'. Further, the nature and extent of skeletal malformations induced were dependent upon the precise stage of somite differentiation at the time when the maternal stress was induced and the extent varied with the degree and the duration of the hypoxia. These carefully documented results clearly justify the authors' conclusion that 'they indicate clearly the importance of minute, and sharply timed, environmental circumstantials in determining variations generally thought to be of genetic origin'. These findings, taken in conjunction with M. J. Pleydell's evidence (*Lancet*, 1957, **i,** 1314) that there may be some association between maternal scarlet fever and mongolism in the child, and the report of J. W. S. Harris and I. P. Ross (*Lancet*, 1956, **i,** 1045) of cleft palate in a baby whose mother was treated with cortisone in early pregnancy, indicate how far we have travelled from the old belief that congenital deformities were predominantly genetic in origin. This, as Ian Donald (*The Practitioner*, 1956, **177,** 389) has pointed out, 'is an encouraging thought and further observations along these lines may help to dispel the prevailing gloom that the majority of fœtal malformations are due to genetic faults and are therefore not preventable'.

Blood Transfusions

To those of us brought up professionally in the days when a blood transfusion was an event and when, to obtain a donor, we often had to go out into the highways and by-ways—or alternatively find one among ourselves—it is difficult to understand what can only be described as the happy-go-lucky way in which transfusions are often ordered nowadays. The demands for blood have now reached a level where, according to the chief medical officer to the Ministry of Health, in his recently published annual report for 1956, 'a further increase in demand may not be met by an equivalent increase in the donor panel, in which case scarcity will follow'. During 1956, 702,593 bottles of blood were issued by the regional transfusion centres, an increase

of about 6 per cent. compared with 1955, and nearly 90 per cent. compared with 1949.

The examples of unnecessary use of blood transfusion, quoted in the chief medical officer's report, indicate the extent to which the National Blood Transfusion Service is being abused: to correct slight degrees of anæmia, to speed convalescence, 'purely as a precautionary measure with no indication'. Some of the trouble is due to the fact that all too often transfusions are ordered by 'the most junior and therefore least experienced staff'. This suggests a degree of carelessness on the part of senior members of hospital staffs that is hardly in keeping with the standards expected of men appointed to such positions. What too many forget is that, not only does this indiscriminate use of blood threaten to deprive a certain number of patients of what may literally be a life-saving measure, it exposes those to whom it is given unnecessarily to a potential hazard—for every blood transfusion means a potential risk to the recipient. It is to be hoped that the profession will now take active steps to ensure that this valuable commodity is not wasted, but is reserved for the use of patients in whom it is indicated.

Leaden Bread

Chronic nephritis is not usually regarded as an epidemic disease, but a recent report from Yugoslavia, by V. Danilovic and his colleagues (*Presse méd.*, 1957, **65**, 2039), suggests that this may occasionally be the case. Their attention was first drawn to the possibility by the high incidence of chronic nephritis in a group of Serbian villages over a period of fifteen years. In one family, for instance, the mother and two daughters had died of the disease within a year, and the father and younger son were suffering from it, leaving only the elder son without any evidence of it. Intensive investigations were carried out on 12 families in one of the affected villages, and these showed that 23 of the 44 members of these families had chronic nephritis. There was no evidence of outbreaks of streptococcal infection in the neighbourhood, nor did any of those affected give a history of infection of the upper respiratory tract.

What finally gave the clue to the solution of this mysterious outbreak was the history some of the patients gave of severe intestinal colic, suggestive of lead poisoning. This clue was followed up, and the authors present a convincing body of evidence to support their final diagnosis of chronic nephritis due to chronic lead poisoning. The source of the lead was as unexpected as the etiological diagnosis. This was the flour from which the local bread was made, and which was found to contain up to 1.6 mg. of lead per kg.—compared with the normal figure of 0.03 to 0.2 mg. per kg. The flour all came from an old mill in the neighbourhood, and examination of its eight grinding stones revealed that these all had a high lead content. The final conclusion therefore was that the flour had become contaminated during the milling process, and that it was in the staple diet of the local inhabitants that had lurked the toxic factor which had produced such disastrous results over the years.

THE CORONARY CIRCULATION IN HEALTH AND DISEASE

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THE coronary arteries were named and described by Galen, but their function in nourishing the heart was probably first appreciated by Harvey (1628). In 1669, Richard Lower published the first account of anastomoses between the coronary arteries. In more recent times full descriptions of the anatomy have been given by Spalteholz (1924) and Gross (1921). Much of our present knowledge of the physiology of the coronary circulation has been reviewed by Gregg (1950).

ANATOMY

The coronary arteries emerge from the aorta just above the aortic valve, arising in the sinuses of Valsalva. They lie at first in the atrio-ventricular groove, the right coronary artery passing to the right to reach the upper end of the posterior interventricular groove where it turns down the postero-inferior surface of the heart to terminate at, or a varying distance above, the apex of the heart. The left coronary artery divides soon after its origin into the circumflex branch (which runs in the atrio-ventricular groove around the left border of the heart) and the anterior descending branch which passes down the anterior interventricular groove to the apex of the heart and sometimes round the apex to the apical part of the posterior inter-ventricular groove. In the human subject the distribution of coronary arteries is subject to considerable variation, which has been studied by Schlesinger (1938, 1940a,b). In about half his subjects the right coronary artery predominated and supplied the whole of the right ventricle, and the posterior half of the left ventricle and septum; in one-third of hearts the circulation was 'balanced', the right coronary artery supplying the right ventricle and posterior half of the ventricular septum, whereas the left supplied the left ventricle and the anterior half of the septum. The least common human pattern is that of left coronary dominance which occurs in about one-sixth of cases; in these the right coronary artery supplies only part of the right ventricle. Schlesinger has claimed that myocardial infarction is most common in hearts with left coronary dominance, and least common in those with a balanced circulation. Gregg (1950) has pointed out that the normal pattern in the dog is left coronary dominance, and much of the experimental work on coronary occlusion has been undertaken in this animal.

The branches of the main coronary arteries which supply the myocardium pass inwards through the muscle from the epicardial to the endocardial surface. They are therefore exposed to the intramural pressure generated

during ventricular systole, and this pressure is greatest in the inner layers of muscle where it is sufficient to prevent and even reverse the flow of blood. The subendocardial layers of the myocardium are therefore normally ischæmic during systole. Myocardial infarcts are commonly predominantly subendocardial and are much more common in the left ventricle where the intramural pressures are higher.

THE GENERAL PATTERN OF THE CORONARY CIRCULATION

Blood emerging from the coronary sinus normally has a lower oxygen content (2 to 6 volumes per cent.) than venous blood returned to the circulation from any other organ, even exercising skeletal muscle. The myocardium thus has the capacity to extract more oxygen from the blood than other tissues. In this respect the coronary circulation can be described as an economical type of circulation, for it demands only a relatively small flow for a given oxygen uptake. This has obvious advantages but it also has the disadvantage that if the oxygen demand of the myocardium increases, this demand can be satisfied only by an increased blood flow. In this respect the coronary circulation differs most widely from the renal circulation, for venous blood returned from the kidney has a high oxygen content, so that the renal blood flow is large compared with the oxygen uptake. The renal circulation is notoriously liable to reduction when the demands of other organs increase, as in exercise, or when the total heart output is restricted, for instance in heart failure (Merrill, 1946). The renal blood flow is largely controlled by factors outside the kidney, and it is often sacrificed to the needs of other organs. This is possible because of its normally low oxygen utilization, which can be increased.

The coronary blood flow, on the other hand, is largely immune to variation by non-cardiac influences, and from moment to moment closely corresponds to variations in the work of the heart. The myocardium stands alone among the tissues of the body in its dependence for survival upon increasing its blood flow in proportion to the work demands placed upon it, and this situation would not be tenable if its blood supply were materially reduced when the demands of other tissues increased, as is the case in the kidney.

FACTORS DETERMINING CORONARY BLOOD FLOW

The factors determining the phases of coronary blood flow.—The blood flow in the coronary arteries differs strikingly from that in peripheral arteries, for it is obstructed during part of systole by the pressure generated in the walls of the ventricles by ventricular contraction. During the early part of ventricular systole, before the aortic valve opens (isometric contraction), the flow rapidly diminishes in the left coronary artery and is soon reversed, but forward flow is resumed during ejection, reaches a maximum just before the peak of systolic pressure in the aorta, and then declines until the aortic valves close, and diastole begins, when it again increases. These variations

are due principally to the relative levels of pressure in the aorta and in the left ventricular muscle, which surrounds the branches of the coronary arteries.

The right coronary artery supplies principally the right ventricle, and the intramural pressure changes in this ventricle are naturally much smaller than in the left ventricle; consequently, although they affect the right coronary flow, there is never any phase of flow reversal. The systolic cessation and reversal of coronary flow in the left ventricle, but not in the right, is of some clinical interest for the majority of myocardial infarcts occur in the left ventricle, whereas the right ventricle is comparatively immune.

The factors influencing total coronary blood flow.—These are of considerable clinical importance and have been fully reviewed by Gregg (1950), who has emphasized the technical difficulties involved in this work, and the fallacies which arise in the interpretation of results. To discuss the experimental observations which have been made and their interpretation is outside the possible scope of this article and the present position can only be summarized. All the following factors have been extensively studied: coronary arterial pressure, coronary venous pressure, asphyxia, anoxia, hypercapnia, myocardial ischæmia, cardiac work and nervous influences. Gregg concludes that the present evidence indicates that the coronary blood supply can, at least in part, be adjusted to the energy requirements of the ventricles. He believes that the major factors which indirectly cause coronary dilatation and increased coronary flow are an increase in ventricular work (increased oxygen demand) and/or a decreased oxygen supply (anoxia).

It is known that the heart has an abundant sympathetic and parasympathetic efferent nerve supply; that both are distributed, *inter alia*, to the coronary arteries, and this implies a probable vasomotor function. On the other hand, experimental stimulation of cardiac nerves does not lead to increased coronary blood flow without, at the same time, evidence of increased cardiac work and, so far, it has not been possible to separate these two possible determinants of coronary blood flow. There is therefore at present no conclusive evidence that the cardiac nerves have a direct vasomotor influence or that they directly control coronary blood flow.

In summary therefore, it appears that the coronary blood flow is controlled by factors within the heart and that it varies with the work load and metabolism of the heart. It is also known that anoxia increases the coronary blood flow, but the precise mechanisms (whether chemical or nervous) by which increased metabolism or decreased oxygen supply augment the coronary flow remain uncertain.

THE ACTION OF DRUGS ON CORONARY BLOOD FLOW

It seems clear that the *nitrites* increase coronary blood flow 'by nearly all methods of study and in nearly all preparations' (Gregg, 1950). This seems to be due to coronary vasodilatation (Boyer and Green, 1941), probably by

the direct action of the drug on the coronary vessels, although indirect action by a change in cardiac metabolism or a decrease in cardiac work cannot be ruled out. The action of *morphine* on the coronary circulation has not been much studied, and its effect on coronary flow is unknown. Nor has the action of *digitalis* been studied by any satisfactory method. There is little satisfactory evidence of the action of atropine, nicotine, or nikethamide. The action of electrolytes (such as potassium) has usually been studied in the fibrillating heart using very high concentrations. There is need for a considerable amount of work in these fields.

PARTIAL CORONARY OCCLUSION

It might be thought that any narrowing of a coronary artery by an atheromatous plaque would reduce the flow of blood, but this is not necessarily so, for it depends upon the peripheral resistance which opposes the outflow from the artery to the tissues. If this is very high, then the proximal narrowing has no effect, for it is merely a comparatively trivial narrowing of a tube which is peripherally much more severely obstructed. In these circumstances, which may exist, as much as ninety per cent. reduction in lumen area may be necessary to reduce flow (Gregg, 1950). If the peripheral resistance is low, however, slight narrowing will reduce flow. In preliminary observations Gregg has found that in the dog the coronary peripheral resistance is relatively high and considerable restriction of the lumen of the main coronary arteries is necessary before inflow decreases. If, however, circumstances arise in which, from increased myocardial work or localized anoxia, peripheral vasodilatation takes place, then an arterial narrowing previously insignificant may seriously limit the blood flow just at the time when the flow requirement is greatest, for the myocardium is largely dependent upon increased flow to meet increased oxygen requirements.

Very significant falls of blood flow are necessary before changes occur in the electrocardiogram, and Visscher (1939) has shown experimentally that they do not occur until the flow is reduced by approximately seventy per cent. Such electrocardiographic changes disappear completely within three to five minutes after reopening the artery.

COMPLETE CORONARY OCCLUSION

Experimentally, sudden death often follows complete coronary occlusion, and in dogs this is usually due to ventricular fibrillation. The liability to ventricular fibrillation and the possibility of controlling it vary considerably from one species to another.

Gregg states that in the dog, sheep and goat, ventricular fibrillation occurs readily and is not spontaneously reversible, but can be stopped artificially. In the frog and turtle it rarely occurs, is hard to induce, and stops spontaneously, whereas in the mouse, rat, cat, rabbit, monkey and fowls although it occurs easily, it recovers spontaneously as a rule.

Although Gregg believes that the human subject resembles the dog in this respect, there are several reports of spontaneous recovery in human ven-

tricular fibrillation. It is widely believed that in man sudden death following coronary occlusion is due to ventricular fibrillation, but Stroud and Feil (1948) showed that if terminal electrocardiograms are taken in fatal cases of coronary occlusion as many die from ventricular standstill (8 of 16) as from ventricular fibrillation. After coronary occlusion, drugs which diminish ventricular excitability (such as quinidine) are as likely to be dangerous as those which might increase excitability (such as digitalis).

THE RELATION OF MYOCARDIAL INFARCTION TO CORONARY OCCLUSION

In the dog permanent occlusion of a main coronary artery almost always leads to myocardial infarction. But even complete occlusion of a main coronary artery will not cause infarction provided the artery is reopened within twenty to thirty minutes (Tennant and Wiggers, 1935; Blumgart, Gilligan and Schlesinger, 1941). Thus, both the duration of reduced blood flow and the degree of coronary occlusion are important in the production of infarction.

When myocardial infarction follows coronary occlusion the ischæmic area does not infarct homogeneously, for areas of surviving muscle remain (Mallory, 1934; Myers, Klein and Stofer, 1948; Hellerstein, 1948; Myers, Klein and Hiratzka, 1949; Wessler, Zoll and Schlesinger, 1952; Prinzmetal *et al.*, 1953; Snow, Morgan Jones and Daber, 1955). If the whole thickness of the ventricular wall became necrotic, then rupture of the ventricle would be inevitable; in fact, a network of living muscle fibres may run through the infarct and, although the area may bulge with each systole, rupture of the ventricular wall is not common, though it does occur.

THE COLLATERAL CIRCULATION

Since strands of muscle survive in an occluded area of myocardium, there must persist, or rapidly develop, some degree of blood supply. Gregg, Thornton and Mautz (1939) showed that, immediately following abrupt occlusion of a main coronary artery in the dog, retrograde coronary blood flow can be measured beyond the obstruction. The blood is bright red and has the same oxygen content as the systemic arteries, and there can be no doubt that some immediate collateral flow is available from unoccluded adjacent coronary arteries, and this flow can be diminished or abolished by clamping other coronary arteries. It was once believed that the coronary arteries were 'end' arteries and did not communicate with each other. In fact, since the experiments of Richard Lower in 1669, it has been known that watery solutions injected into one coronary artery could be recovered from the other. It was believed, however, that these anastomoses between the coronary arteries were too small to be functionally useful.

The function of these channels in the hours and days following infarction has been clearly demonstrated in the dog by Gregg and his associates (Gregg, 1950, 1952). Although the retrograde flow is very slow in the

first few hours after coronary occlusion, it soon begins to increase, has doubled in forty-eight hours, and is three or four times the initial level within a week. At this time the muscle fibres in the infarcted area, which at first bulged with each systole, begin to show systolic shortening. Within about four weeks the retrograde flow approximates to the normal inflow, and the collateral pressure pulse may approximate to the normal in pattern and magnitude. It is not known whether this collateral circulation is established solely by the enlargement of pre-existing channels or whether new channels develop. The slow development of collateral coronary circulation is in striking contrast to that in the femoral and carotid arteries, in which a large collateral circulation can develop within one to two days (Eckstein, Gregg and Pritchard, 1941). Yet much of the myocardium may survive in an occluded area, presumably owing to the capacity of the myocardial muscle to extract a large proportion of the oxygen carried to it in the blood.

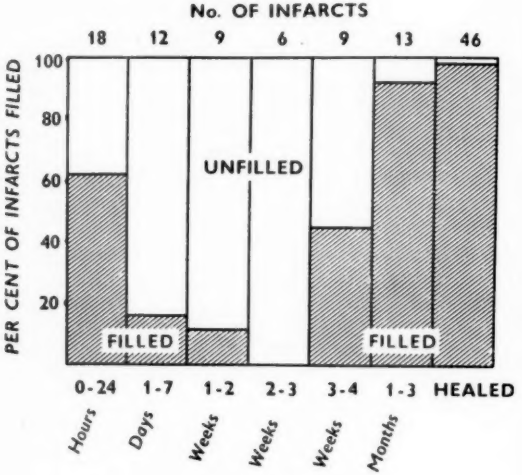

FIG. 1.—This diagram summarizes the data obtained by injecting hearts containing 113 cardiac infarcts with radio-opaque injection media. Apparently normal filling is found in almost all infarcts histologically estimated as over four weeks old. The filling of recent infarcts in some cases is not yet explained. The technique used is described by Snow, Morgan Jones and Daber (1955).

THE HUMAN CORONARY COLLATERAL CIRCULATION

In 1940, Blumgart, Schlesinger and Davis demonstrated very clearly revascularization of the human myocardium following coronary occlusion. They perfused the right and left coronary arteries with differently coloured injection media and showed that apparently complete filling of an occluded area of myocardium in the field of supply of one coronary artery took place from the other coronary artery. The observations of Snow, Morgan Jones and Daber (1955) on the collateral filling of infarcted areas in man have shown that, on post-mortem injection at physiological pressures, the degree of collateral filling increases with the time after infarction, and the vascular pattern becomes apparently normal in almost all infarcts more than four

weeks old (fig. 1). Although it is not possible to obtain precise data in the human subject, comparable to the experimental evidence which has been collected in the dog by Gregg and his associates, the available data indicate that the general pattern of myocardial revascularization is similar in the dog and man.

Although there is little doubt that the principal pathway of revascularization is from unoccluded arteries distal to the occlusion, there is some evidence that other intracardiac (Gregg, 1950) and extracardiac (Burchell, 1940) pathways may play a part. In addition, it is known that coronary artery occlusions usually recanalize, and may ultimately carry a significant blood supply, for further occlusions of recanalized arteries may lead to new areas of infarction (Snow, Morgan Jones and Daber, 1955).

THE EFFECT OF EXERCISE AND CORONARY ARTERIAL NARROWING UPON COLLATERAL VASCULARIZATION

Eckstein (1957) has shown that a moderate degree of coronary arterial narrowing in the circumflex artery of the dog will induce collateral vascularization, and that the amount of the collateral circulation is proportional to the degree of narrowing. If the animal is exercised the amount of collateral vascularization is increased. It might be dangerous to imply that exercise is a useful agent for promoting collateral vascularization after acute coronary occlusion, for in these circumstances survival of the ischæmic myocardium might be imperilled. It is more reasonable, however, to suggest, with Eckstein, that when chronic narrowing of the coronary arteries is present, judicious exercise may be beneficial, though it may be difficult to know what amount of exercise is judicious in the circumstances. These results are certainly an encouragement to regular moderate exercise for asymptomatic people in the coronary age-group, with or without known coronary disease, and even for those with mild angina of effort, provided the prescribed exertion falls short of provoking pain. They may discourage the idea that rest is beneficial in quiescent coronary disease.

THE SEQUENCE OF EVENTS FOLLOWING CORONARY OCCLUSION

Following coronary occlusion an area of myocardial ischæmia is created which is likely to be least intense near the periphery of this area, where it is nearest to the branches of other coronary arteries. There is at first only a very small collateral blood supply but, owing to the ability of cardiac muscle to extract a large proportion of the oxygen from its blood supply, some of the ischæmic muscle survives. This cannot, however, contract effectively, for the ischæmic area bulges with each systole. Histologically, throughout the area there is a network of surviving myocardium. There is evidence to indicate that parts of this surviving muscle may subsequently die without any additional arterial occlusion (Snow, Morgan Jones and Daber, 1955) and this is naturally most likely to occur near the centre of the ischæmic muscle.

This liability to extension of the infarcted area exists for about four weeks. All the evidence, in animals and in man, indicates that by this time collateral vascularization is considerable in the majority of cases. So, for about four weeks following coronary artery occlusion, necrosis may extend from the centre, and revascularization grows from the periphery; the ischæmic muscle is gradually absorbed by one or other of these processes and finally well-revascularized muscle is sharply divided from a circumscribed scar much smaller than the original ischæmic area. This is the meaning of 'healing' after coronary thrombosis.

This picture is probably substantially correct, for much of it is based upon well-founded physiological and histological observations and it can, to a considerable extent, be confirmed by careful clinical observation of the course of myocardial infarction and by the changes in serial electrocardiograms, in which the regression of ischæmia is marked by improvement in T wave changes, and the development of new areas of infarction may be recognized by the abolition of previously persisting R waves. There is no doubt, however, that improvement in T wave abnormalities may continue for a much longer period than four to six weeks, and there may be improvement up to a year after the onset of infarction. This is an aspect of healing in human myocardial infarction which deserves further study.

I am indebted to Dr. P. J. D. Snow for permission to use fig. 1 from his M.D. thesis.

References

Blumgart, H. L., Gilligan, D. K., and Schlesinger, M. J. (1941): *Amer. Heart J.*, **22,** 374.
——, Schlesinger, M. J., and Davis, D. (1940): *Ibid.*, **19,** 1.
Boyer, N. H., and Green, H. D. (1941): *Ibid.*, **21,** 199.
Burchell, H. B. (1940): *Arch. intern. Med.*, **65,** 240.
Eckstein, R. W. (1957): *Circulat. Res.*, **5,** 230.
——, Gregg, D. E., and Pritchard, W. H. (1941): *Amer. J. Physiol.*, **132,** 351.
Gregg, D. E. (1950): 'Coronary Circulation in Health and Disease', London.
—— (1952): 'Aortic and Coronary Flow', Ciba Foundation Symposium, London.
——, Thornton, J. J., and Mautz, F. R. (1939): *Amer. J. Physiol.*, **127,** 161.
Gross, L. (1921): 'The Blood Supply to the Heart', London.
Harvey, W. (1628): 'De Motu Cordis', Frankfurt.
Hellerstein, H. K. (1948): *Amer. Heart J.*, **36,** 535.
Lower, R. (1669): 'Tractatus de Corde', London.
Mallory, T. B. (1934): *New Engl. J. Med.*, **210,** 42.
Merrill, A. J. (1946): *J. clin. Invest.*, **25,** 389.
Myers, G. B., Klein, H. A., and Hiratzka (1949): *Amer. Heart J.*, **38,** 547.
——, ——, and Stofer, B. E. (1948): *Ibid.*, **36,** 535.
Prinzmetal, M., *et al.* (1953): *Circulation*, **7,** 1.
Schlesinger, M. J. (1938): *Amer. Heart J.*, **15,** 528.
—— (1940a): 'Blood, Heart and Circulation', A.A.A.S., Publ. No. 13, 61.
—— (1940b): *Arch. Path. (Chicago)*, **30,** 403.
Snow, P. J. D. (1955): M.D. Thesis, University of Manchester.
——, Jones, A. Morgan, and Daber, K. S. (1955): *Brit. Heart J.*, **17,** 503.
Spalteholz, W. (1924): 'Die Arterien der Herzwand', Leipzig.
Stroud, M. W., and Feil, H. S. (1948): *Amer. Heart J.*, **35,** 910.
Tennant, R., and Wiggers, C. J. (1935): *Amer. J. Physiol.*, **112,** 351.
Visscher, M. B. (1939): *J. Amer. med. Ass.*, **113,** 987.
Wessler, S., Zoll, P. M., and Schlesinger, M. J. (1952): *Circulation*, **6,** 334.

ANGINA PECTORIS

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ANGINA pectoris is essentially a symptom indicating a discrepancy between the work of the myocardium and the available supply of oxygen. The common cause of an inadequate blood supply is occlusive disease of the coronary arteries, usually due to atheroma, sometimes luetic coronary ostial stenosis, and, very rarely, coronary embolism or congenital abnormality of the coronary vessels. Anæmia causes an inadequate supply of oxygen and is thus an additional adverse factor in occlusive coronary disease; pain rarely results from anæmia alone. The decrease in available oxygen supply necessary to produce angina may be relative when a normal or slightly reduced supply must serve the greatly increased muscle mass of ventricular hypertrophy. When angina pectoris is associated with endocrine disease, such as myxœdema and diabetes mellitus, or with xanthomatosis, it is due to coronary atheroma.

THE PAIN SYNDROME

The pain is usually over the upper sternum and tends to have a transverse rather than a vertical distribution. The area may be extensive but sometimes it is small; it spreads centrifugally to the base of the neck, jaws, shoulders, arms and even hands. Occasionally it is first felt in the arm or jaw. Often, patients will deny pain in the arms but complain of a curious sense of numbness, weakness or heaviness there. Pain sometimes spreads across the shoulders and over the upper thoracic vertebræ, radiating to the sides of the chest. Many describe the pain as 'going through to the back'. It is characteristically pressing, squeezing, constricting, or vice-like, never pricking or stabbing, although such words may be used through inadequate diction.

It is in the essence of the syndrome that cardiac pain is produced by increased cardiac work, which is commonly caused by effort. It is not always realized that the sustained effort of walking is more provocative than a short burst of physical effort. Patients are not infrequently surprised by the absence of pain during a day's work which may involve short periods of quite strenuous effort.

The threshold of pain production is lowered by any factors causing more cardiac work, such as an increase of pace, walking up an incline, walking against the wind, effort after a meal or increased mental tension. When other factors are absent it is common for the pain to be induced by constant amounts of effort. The pain persists for as long as effort is tolerated and it is therefore relieved by rest in one to five minutes. Sometimes a vague discomfort or soreness persists. In some rare mild cases relief may come in spite of continued effort but in all severe cases pain brings the patient to a standstill. In advanced cases even the smallest effort may provoke pain.

During attacks of pain there may be no signs but often there are facial pallor and a rather fixed anxious expression.

THE ELECTROCARDIOGRAM

The electrocardiogram is a necessary investigation in all suspected cases of angina pectoris. It provides important evidence of the existence of muscle damage and in those cases in which damage is slight the pathological changes in the electrocardiogram appear after exercise. The incidence of an abnormal electrocardiogram in patients with angina depends upon the comprehensiveness of the investigation and the skill of the interpreter; thus, there is no agreement on this point but it is probable that some 85 per cent. of patients show some abnormality. There may be gross changes indicating previous extensive infarction of the muscle, but more often the changes are slight and confined to the S-T interval and T wave. The electrocardiogram recorded at rest may be physiological. In such cases the test should be repeated after either a standard amount of exercise or after just enough effort to cause discomfort; the functional injury caused by effort is sufficient to produce transient abnormalities in the S-T interval and T wave. Even after these procedures some 5 per cent. of electrocardiograms remain normal early in the disease.

DIAGNOSIS

The recognition of angina pectoris depends essentially upon the patient's ability to describe his subjective sensation and the physician's ability to evaluate the situation. Mostly the diagnosis can be made with assurance on the history. The language of sign and gesture is of great value. The clenched fists across the chest, the open hands gripping the side of the chest, a right hand gripping the left wrist or biceps are often of greater significance than the spoken word. Direct questions, albeit posed obliquely or inversely, should be used and certainly not avoided. Few patients talk of other symptoms when giving a history of angina; their concern is with the one pain. Most will remember the circumstances and time of the first attack.

The response to nitroglycerin may be misleading, for the drug relieves spasm of smooth muscle generally and psychoneurotics may also be relieved of symptoms for a time. A diagnosis of angina pectoris, the symptom, is insufficient; cardiac causes other than coronary atheroma should be sought and excluded. In all cases the electrocardiogram is a necessary investigation. Abnormalities not only confirm the diagnosis but aid in assessing the degree of myocardial damage.

DIFFERENTIAL DIAGNOSIS

Few of the many causes of chest pain should be confused with angina if a detailed history has been obtained and an adequate electrocardiogram has been recorded. Electrocardiography shows a higher degree of positive correlation with the underlying cause than many other investigations in medicine.

Neurotic patients often have numerous symptoms and the pain is usually beneath the left nipple; occasionally it is over the sternum and related to

effort, but the relationship is usually inconstant or the pain lasts for hours and comes on after the effort is over. The electrocardiogram is then normal before and after exercise.

Pain arising from other structures in the mediastinum or midline may cause difficulty. *Œsophageal pain* is rarely related to effort although it may have the same quality and distribution as angina pectoris. *Hiatus hernia* sometimes produces a similar spasmodic pain, but there is no relation to effort although a particular posture or movement may produce the pain. Most patients with hiatus hernia are asymptomatic and in others dyspepsia is the main complaint. Hiatus hernia is not uncommonly associated with angina pectoris; both conditions occur with increasing frequency after middle life. The diagnosis of œsophageal spasm, œsophageal arrhythmia and hiatus hernia is confirmed by radiological investigation.

Pain referred from *musculo-skeletal lesions* may superficially resemble angina pectoris when movement is confused with effort: *cervical spondylosis* may cause pain over the upper chest and pain in the arms. Lesions of the upper dorsal spine may likewise cause neuritic pain in the front of the chest and a sudden movement of the chest or sneezing may produce it. *Gall-bladder disease* is said to cause confusion with angina pectoris but it rarely does so; these conditions sometimes coexist and, whilst in some way gall-bladder disease may lower the threshold for cardiac pain when there is coronary disease, it does not alone produce chest pain related to effort. When a clear history of angina of effort is obtained then, in my experience, there is rarely any problem of differential diagnosis: even if radiology shows a hiatus hernia, or gall-stones or bronchial carcinoma, the subject probably has both conditions.

TREATMENT

The treatment of individual attacks of angina is well known. Sufferers soon discover that relief comes with rest. Patients should be advised to reduce or stop exertion at the first suggestion of pain. No drug has superseded the nitrites since their beneficial action was described by Lauder Brunton. Glyceryl trinitrate (trinitrin) is the drug of choice of the many nitrites which are available: the tablets should be fresh and kept in an air-tight container. Trinitrin tablets act rapidly when chewed; they should neither be taken as a regular medication nor swallowed. The drug is non-toxic and excreted rapidly so that no restriction should be placed on their use if attacks are very frequent. Tablets containing 1/100 grain (0.6 mg.) are mostly required but some patients find that they can manage with half a tablet and others require two or three for severe attacks. The procedure of breaking capsules of amyl nitrite and inhaling the unpleasant vapour is best avoided. Although amyl nitrite has a slightly more rapid action than trinitrin only a few patients prefer this drug. Trinitrin should be used to prevent attacks when a constant amount of effort is known to cause the pain.

The assessment of any *long-term treatment* in angina pectoris is extremely difficult. It is not always realized that myocardial ischæmia shows an

inherent tendency to improve over long periods of time, especially in patients who are able to adjust their minds and life situation to the disease. Many observers have noted that when a patient is started on any new form of treatment improvement is almost invariably the rule. The conclusion cannot be escaped that psychological factors as well as many physical ones are of importance. An understanding doctor-patient relationship is as important in this disease as in any condition in the whole field of medical practice.

A reasonable explanation of the cause of the pain, tinged with optimism, is essential for the educated patient. All should be advised to avoid those situations which tend to cause angina. Effort should not be avoided altogether but rather encouraged, although confined to levels below that which provokes pain. Exercise should not be taken after meals. Rush should be eliminated, situations charged with emotional tension should be avoided and an attitude of quiet acceptance of the life situation should be developed —easier said than done—but with help from an understanding physician an attitude of mental relaxation may be achieved. Obese patients should be advised to reduce weight by means of a low-fat regime, but the value of weight reduction has probably been exaggerated and too vigorous methods may cause weakness and anxiety. The work situation often needs some adjustment: in most cases it is better to remain in gainful employment.

Two or three weeks of modified bed rest or an armchair regime are advisable for patients soon after the onset of angina. Bed rest and anticoagulant therapy are indicated when the severity and frequency of the attacks are increasing rapidly, for this points to an impending episode of salient infarction. Sedatives should be given to patients who are excessively anxious and in whom emotional factors readily produce pain: chlorpromazine is sometimes beneficial in such patients.

Tobacco smoking may be harmful but few patients find relief when the habit is dropped. *Alcohol* is not contraindicated and its tranquillizing effects are probably beneficial.

Anti-anginal drugs.—When frequent severe attacks are causing much disability various drugs may be tried. Most of these will produce some initial improvement, depending upon the mental receptiveness of the patient and his physician's ability to reassure and convince rather than on any pharmacological action of the drug. Pentaerythritol tetranitrate is the most favoured but in carefully controlled trials by double-blind techniques neither patient nor doctor is readily able to distinguish drug from placebo by the relief of symptoms. Khellin has been used but the results are not impressive after the inevitable initial improvement. When nocturnal attacks of pain are troublesome a suppository of aminophylline may bring relief and uninterrupted sleep. Digitalis should be used for symptoms suggesting the onset of failure but it is not indicated in the absence of failure.

The production of *hypothyroidism* by anti-thyroid drugs is worth a trial in seriously disabled patients, but has disadvantages: hypothyroidism is unpleasant and many patients prefer their angina to distressing mental lethargy.

Anticoagulant therapy is indicated when there is a crescendo history of angina or when brief attacks of pain at rest suggest an impending attack of cardiac infarction. The evaluation of long-term anticoagulant therapy in coronary disease awaits the results of carefully controlled trials over long periods: it is probable that the frequency and severity of episodes of infarction may be diminished but the effect of such treatment on effort pain is not impressive. Bi-weekly injections of heparin have been advocated but trials have not shown a consistent improvement in the symptoms.

SURGICAL TREATMENT

Surgical methods of treatment have been developed during the past three decades. The aim has been either to cut pain pathways or to improve the blood supply of the myocardium. Afferent pain fibres reach the spinal cord in sympathetic nerves of the upper dorsal region and it follows that cardiac pain may be relieved by high dorsal sympathectomy; at least the first four thoracic ganglia should be removed for the best results. Neither sympathectomy, stellate ganglionectomy nor posterior rhizotomy, however, has gained general acceptance: such operations carry considerable risk, improvement may be only temporary, oppressive sensations and weakness on effort are sometimes as unpleasant as angina and in any case it may be harmful to remove the warning of impending ischæmia.

Operations designed to improve blood supply are of two types: those directed to increasing the supply of blood from outside the heart and those aimed at increasing intercoronary anastomosis. The first method includes attempts to create a vascular adhesive pericarditis: cardio-omental anastomosis and implantation of the internal mammary artery. The second method includes partial or complete occlusion of the coronary sinus and its anastomosis to the aorta. The more elaborate operations combine both methods. The operative mortality is fairly high but some good results have been claimed. Much longer periods of controlled observation are necessary, however, before the results of these ambitious techniques can be assessed. It is probable that they will belong to a brief phase in the history of therapeutics.

THE COURSE OF ANGINA PECTORIS

One of the features of this disease is its capricious uncertain course, but in the past too much emphasis has been placed on its malignancy and it has been too readily associated, particularly in the lay mind, with sudden death. Attacks may subside in severity and even completely disappear for long periods. In most patients, however, the malady tends to pursue a progressive but episodic downhill course. The average duration of life is eight years but many live for much longer. Myocardial infarction is the great danger but even after this, cardiac ischæmia shows an inherent tendency to spontaneous improvement. Under medical treatment and with moderate adjustment of physical and mental activities most patients may be returned to a useful life for long periods.

MYOCARDIAL INFARCTION

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THE pathogenesis of myocardial infarction has been fully dealt with elsewhere in this symposium, but two points are worth emphasis. Whilst myocardial infarction is generally the result of occlusion of a coronary artery, usually by thrombosis, there is a proportion of cases in which no such occlusion is demonstrable at necropsy. It is clear that to label all cases of myocardial infarction as 'coronary thrombosis' is misleading. Secondly, whilst it is well recognized that in contrast to anginal seizures the onset of myocardial infarction generally bears no relation to effort, in my experience there are some cases with a clear-cut relation of an attack to an immediately antecedent strenuous act of a type to which the patient has not been accustomed: e.g. cranking a car on a frosty day; cutting down a tree in the garden; or running with a heavy suitcase to catch a train. The point is important since current teaching is against such a relationship and authority to that effect is often quoted in legal arguments. So long as thrombosis is assumed to be the cause of all infarcts the current view of non-relation to activity is perhaps tenable: but infarction without occlusion could clearly result from myocardial overload. Middle-aged men of sedentary habit indulge in sudden strenuous unaccustomed exercise at some peril.

CLINICAL FEATURES

In a proportion of cases, probably around 20 per cent., the onset of the major episode is heralded over a few days by anginal pain: either a new experience for a healthy man, or a sudden exacerbation in frequency and severity of attacks in a known anginal subject. Attacks produced by trivial exertion, or lasting longer than the usual effort angina, e.g. up to twenty to thirty minutes, are common prodromata of a major infarction.

The classical attack is too well known to require elaboration: the syndrome of pain, which in site, radiation and nature is identical with angina but which is commonly very severe and typically persistent over several hours, accompanied, it may be, by dyspnœa, anxiety, restlessness, and, in severe cases, by shock, is familiar to every practitioner in the western world. The subsequent course over the next few days, with low fever, raised white blood cell count and erythrocyte sedimentation rate, with falling blood pressure and faint heart sounds and, in about 15 per cent. of cases, with a transient pericardial friction rub, is also familiar. It is the atypical cases which merit further description, for in those the diagnosis may be difficult.

In some patients *the pain is atypical in site*, being felt maximally or solely in the jaw or tongue, between the shoulder blades, in the arms, or in the

upper abdomen: i.e. in one of the peripheral areas to which radiation of central sternal pain usually occurs.

Some patients, who may later be proved to have sustained a substantial infarct, have relatively *brief and mild pain*, commonly attributed by patient and doctor alike to 'indigestion'. The occurrence at night, the temporary relief from belching wind, and the transient nature of the attack combine to

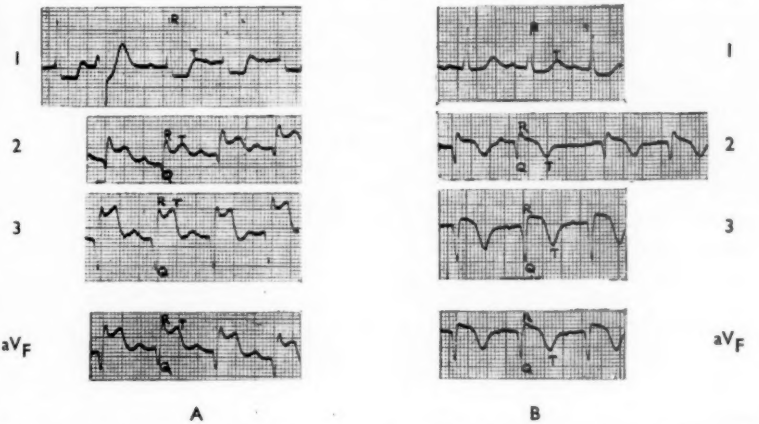


FIG. 1.—In record A taken within a few hours of onset of infarction, there is gross depression of RT segment in lead I with corresponding elevation of RT in leads II and III. These leads show deep Q waves. Both reflect the pattern of the left leg lead (aVF). In tracing B, taken two days later, the RT shifts have largely subsided and deep inversion of T is developing in leads II, III, and aVF. The ECG is typical of posterior infarction.

mislead the unwary. Again, in some patients pain may be either absent or quite overshadowed by *dyspnœa*, so that the case presents as a nocturnal paroxysmal dyspnœa (cardiac asthma), and the underlying infarct is missed. *Syncope* may be the initial symptom, with or without subsequent pain.

Disturbances of cardiac rhythm are common in myocardial infarction, and can prove troublesome in diagnosis and management. Of these, extrasystoles are the commonest: when extrasystoles are few and infrequent, they are of little significance. When, however, they occur in numbers, several each minute, possibly with periods of bigeminy or runs of successive ectopic beats, they are of some import: they may herald a dangerous paroxysm of ventricular tachycardia or the onset of fatal ventricular fibrillation. Atrial (auricular) fibrillation is a common finding, and may be paroxysmal or permanent. If the ventricular rate is high the serious effects on an already damaged heart muscle need no emphasis. A ventricular tachycardia at high rate (180 to 210 per minute) may dominate the clinical picture, and a difficult diagnostic quandary arises—for the fast rhythm may be produced by myocardial infarction, whilst in some patients without infarction, attacks of paroxysmal tachycardia lead to persistent pain from poor diastolic filling

and consequent impaired coronary blood flow. In some patients with infarction of the diaphragmatic surface of the heart ('posterior' infarcts) the blood supply to the AV bundle is impaired, and heart block, partial or complete, results; this is generally temporary, clearing in a few days.

In general, such arrhythmias influence the prognosis unfavourably in any given case.

FIG. 2.—Three records by the same lead from one patient, to show the early QT elevation subsiding with the development of deep T inversion. The deep Q waves remain unchanged and are lasting evidence of underlying transmural infarction.

Shock.—Some degree of shock is the rule in myocardial infarction, and with extensive infarction shock may be profound. In these cases the patient may be ashen-grey, restless, cold, clammy, with fast small pulse and low blood pressure. Pressures of 80/50 mm. Hg are not uncommon; in some the blood pressure cannct be recorded. Spontaneously or with treatment some recover in a few hours, but the mortality in this group is formidable.

ELECTROCARDIOGRAPHIC CHANGES

This is not the place for discussion of the minutiæ of the electrocardiographic (ECG) diagnosis. Certain broad principles, however, can be outlined, and the limitations and fallacies of the technique discussed.

The occurrence of myocardial infarction sets in train two sets of ECG changes which are fundamentally different in causation and course. The acute damage to living muscle cells sets up injury currents, monophasic deflections of the type familiar from physiological work, which deform the portion of the ECG curve between RS and the T waves. The ST segment, normally isoelectric, is displaced several millimetres upward or downward, the displacement being opposite in sign in leads I and III (if upward in lead I, downward in lead III; and conversely) (fig. 1). In leads from the chest wall ST is displaced upward or downward according to the site of the infarct. These 'ST shifts' are seen shortly after the onset, persist for some hours or a day or two, and disappear, to be followed later by gross deformity of the T waves. In general, leads which have shown upward ST shifts develop deep sharp inversion of T in the succeeding days and this may persist for many weeks (fig. 2).

The second set of ECG changes is due to cessation of activation (and therefore of depolarization) of killed muscle cells. If a sufficient area of myocardium is necrotic, the initial deflections (QRS) in standard leads are altered, and leads from points on the chest wall overlying the infarcted area are grossly changed. In effect, the ECG from such points reflects simply the potential changes of the underlying ventricular cavity, and in

place of a complex positive-negative (RS) deflection shows simply a deep negative (Q) wave. Anterior infarcts are thus best detected in chest leads, and their extent can be gauged from the number of leads so affected. So-called 'posterior' infarcts, affecting the diaphragmatic surface, show a similar Q in the left leg lead, and in standard leads II and III which incorporate the left leg in their connexions. Various ECG patterns have been identified with infarction of specific areas of the heart muscle as in the examples figured (fig. 1, 3 and 4).

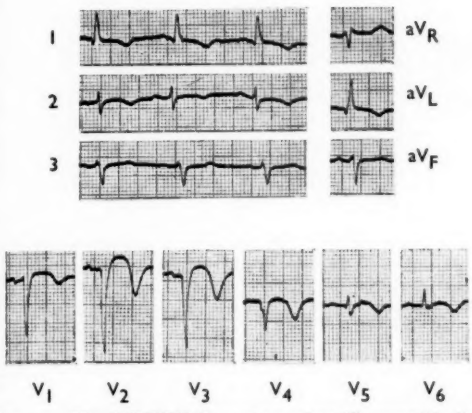


FIG. 3.—The full ECG from the patient illustrated in fig. 2. Q is the sole initial deflection in chest leads V1, V2, V3 and V4. The infarct is antero-septal in type and transmural.

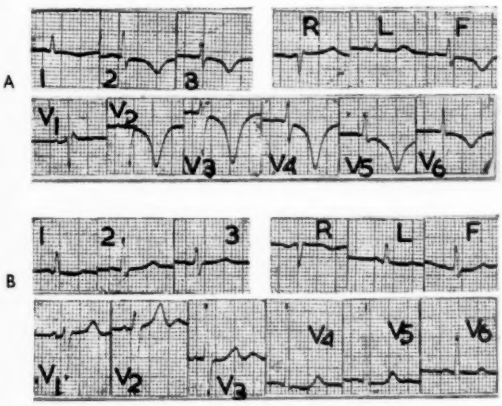
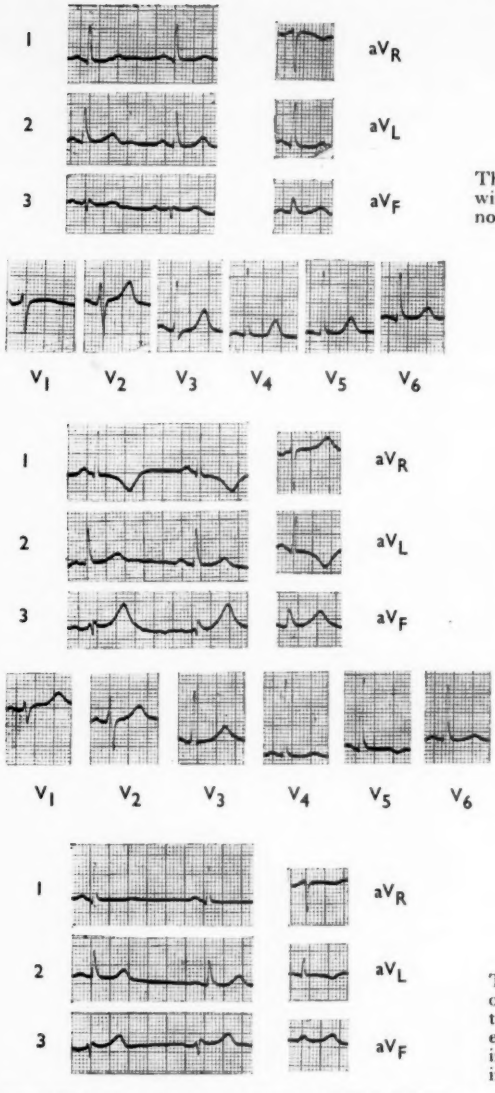


FIG. 4.—(A) ECG a few days after clinical onset of myocardial infarction. Gross T wave changes of type associated with subendocardial infarct. (B) Five weeks later gross changes have subsided; ECG no longer characteristic.

These QRS changes are more permanent than the ST-T changes described above and in many patients remain for life as tell-tale evidence of old infarction.

It should be noted that the ECG pattern in myocardial infarction at first changes from day to day, and later from week to week. It is the sequence of changes that is diagnostic in many cases, especially when the original ECG is atypical. Again, the ECG changes, whilst commonly already present one to two hours after the incident, may be delayed in appearance even up to twenty-four to forty-eight hours, so that a single early 'normal' ECG can be misleading. Conversely, when ECG changes are early and transient (ST-T pattern without QRS changes), a single ECG after three to four days may show no abnormality and again may be misleading. This is particularly liable to occur when the infarct has been small, affecting a few patches of muscle deep in the ventricular wall: a condition associated clinically with relatively brief pain of moderate



The original EGG taken within 3 hours of onset is not significantly abnormal.

The record 24 hours later, showing appearance of deeply inverted T waves in leads I and aVL, with distorted abnormal upright T in leads III, aVR and aVF. There is shallow inversion of T in V5. and the T waves in V4 and V6 are flattened.

The record 17 days after onset. The EGG has practically returned to normal except for flattening of T in lead I and T inversion in AVL.

FIG. 5.—Three electrocardiograms from one patient who presented with a clear-cut clinical picture of myocardial infarction.

severity and not associated with shock ('acute coronary insufficiency') (fig. 5).

It is a dangerous misconception to believe that the ECG always reveals infarction when such is present. Even with multiple lead techniques there are undoubtedly a few cases in which normal ECGs are recorded—it may be on repeated occasions—and when there is no doubt from the clinical features and subsequent course that infarction has occurred. When clinical judgment conflicts with instrumental evidence, the sound rule here, as in general in medicine, is to trust the clinical findings.

DIFFERENTIAL DIAGNOSIS

The classical case is unlikely to be mistaken for anything else, but cases are common in which atypical features may readily lead to a confusion that may be hard to resolve. Such, for example, are the cases in which pain is subordinate to dyspnœa or syncope, or in which the patient presents with embolic complications (hemiplegia; limb gangrene), having failed to seek medical advice for the antecedent coronary episode. The site of the pain may be misleading. High central upper abdominal pain with shock may simulate a perforated peptic ulcer or biliary colic; conversely, patients suffering from acute hæmorrhagic pancreatitis may be diagnosed as myocardial infarction. The pain of pulmonary infarction is usually referred to one or other side, but the initial shock and dyspnœa of a massive pulmonary embolism can be difficult to differentiate. Central chest pain rarely leads to confusion but on occasion acute non-specific pericarditis may be misdiagnosed as the more serious lesion. In the former, friction is audible early, even on the first day, shock is absent, and the ECG pattern of ST shifts is 'concordant': i.e. upwards in all three standard leads, whilst QRS changes are absent. Dissecting aneurysm may closely simulate infarction. Changing site of maximal pain, disappearance of pulse at wrist or groins, equivocal ECG and x-ray findings are all of value in the diagnosis.

A condition with no relation to the cardiovascular system which commonly simulates myocardial infarction is hiatus hernia, which may give rise to persistent substernal pain, commonly at night in recumbent subjects. The absence of shock with prolonged pain, the relief of pain by sitting up or standing and its recurrence on lying down, the absence of ECG changes and the course, all give clues which justify radiological examination, which may clinch the diagnosis. Unhappily, some patients have both a myocardial infarct and hiatus hernia!

COURSE AND PROGNOSIS

Myocardial infarction is a dangerous lesion and carries considerable risk to life. The highest mortality occurs in the first twenty-four hours after onset. Many victims die at once—an unknown proportion of the total since they never reach hospital and even at a coroner's post-mortem examination no myocardial lesion may be detected—understandably, since the affected area and the remainder of the myocardium in these cases die simultaneously. Among those surviving long enough to reach hospital, one-third of all the

deaths occur in the first twenty-four hours, and one-half in the first week. Thereafter the risks are less, and diminish steadily over the next six weeks. The early deaths are due to shock, ventricular fibrillation, or recurrent infarction. The later deaths are from left ventricular failure, congestive failure, embolism, or cardiac rupture, with a proportion due to recurrent infarction and its attendant hazards.

The over-all case mortality in the first six weeks after the onset is around 25 to 30 per cent. The figure shows considerable variation in different hospitals: variations not due solely (or mainly) to efficacy of treatment, but influenced by many factors. Chief of these variables are first the attitude of general practitioners in respect of hospital cases, whether all cases, slight and severe, are sent for admission or only the seriously ill, and secondly the average interval from onset till admission. If cases are sent in on the second or later days, automatically the hospital death rate is lower since so many severe cases die in the first twenty-four hours—i.e. before admission. Comparison of series of cases and consequently evaluation of methods of treatment are fraught with difficulties.

The prognosis for the individual patient must always remain uncertain. Some desperately ill patients make an unexpected and straightforward recovery, whilst others, apparently with little upset and relatively trivial infarcts, as unexpectedly succumb. In general, however, certain factors have a recognized adverse effect on the chances of survival—advanced age, a history of previous infarcts or of failure, severe shock, long-continued or recurrent pain, the occurrence of arrhythmias, or embolic incidents. Infarction in really young people (under 30 years) carries a high death rate, although in my own experience a man of 22 and a pregnant woman of the same age ran uneventful courses to recovery.

The *late prognosis* of myocardial infarction is known from follow-up studies. These show that of those who survive the initial attack, a certain number die within a few months, others during the first year, and a gradually diminishing number during each subsequent year. The risks are greatest in the early months, or in the first year, and those who survive for three or four years have a relatively good life expectancy. Recurrence of infarction and the development of heart failure are the main hazards. The latter is particularly common in patients who have sustained several infarctions, and whose myocardium is correspondingly widely scarred. Angina of effort is a common sequel to infarction. These are estimates of group prognosis. Individual prognosis is highly variable, and the outcome for any individual patient largely unpredictable. It should be remembered that, whilst some patients succumb soon after the first attack, many live active unhampered lives for many years. In general, patients with little residual disability after their attack have the best prognosis. In my experience many patients have survived a decade, and some much longer.

TREATMENT

The immediate indication in treatment is to relieve pain, for which morphine,

15 to 30 mg. ($\frac{1}{4}$ to $\frac{1}{2}$ grain), can be given hypodermically. If shock is severe, absorption may be delayed, and in such cases it is wiser to give a smaller dose (8 to 10 mg. [$\frac{1}{8}$ to $\frac{1}{6}$ grain]) intravenously. In my opinion, once a firm diagnosis is reached on clinical grounds, the general practitioner should give an initial dose of heparin, 100 mg. (10,000 units) intravenously.

It is wise to send patients to hospital as soon as possible. Very shocked

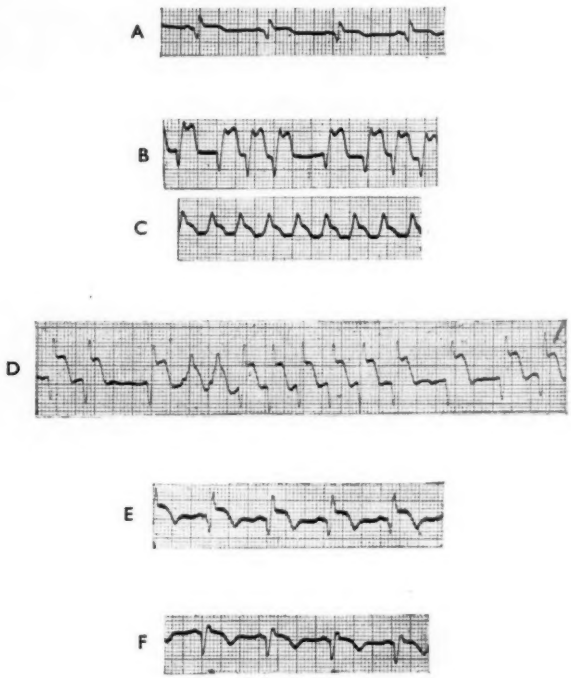


FIG. 6.—In the early course of his illness this patient showed various arrhythmias which settled down to sinus rhythm (A). A recurrence of sudden severe chest pain was associated with gross irregular tachycardia. The ECG (B) shows atrial fibrillation, rate 160/min., with considerable RT displacement. While the record was being taken the rhythm changed (C) to ventricular paroxysmal tachycardia, rate 200/min. Within 15 minutes the rhythm reverted to atrial fibrillation (D). Quinidine by mouth was followed by resumption of normal rhythm, shown in (E) taken 2½ hours later. In (F), taken next day, the RT shift has practically disappeared.

It would appear that the pain and RT distortions in this episode were due to extremely rapid heart action and not to fresh myocardial infarction.

patients may be too ill to move, and should not be disturbed, particularly as their outlook is so grave. But in general it is only in hospital that the nursing care and complete rest so essential in this condition are available. Be the wife ever so devoted, the task of nursing a heavily built, middle-aged, prostrate husband is likely to be beyond her strength. Further, in hospital

all facilities for treatment are available, including those for control of anticoagulant treatment.

It is profitable to consider the hazards run by the patient as a guide to what one can do to avert catastrophe, and as an index of probable efficiency of treatment. Of the early hazards, *severe shock* occurs in patients with large infarcts, is a grave prognostic sign, and is commonly resistant to therapy. Treatment along general lines—warmth, rest, sedation—should be supplemented by oxygen, given freely by mask or nasal catheter at a rate of 6 to 8 L./minute. Attempts to raise the blood pressure by intravenous infusion (e.g., dextran; hypertonic glucose) are prone to lead to circulatory overload and failure. Pressor drugs may be used, for example methylamphetamine given intramuscularly, but adrenaline should be avoided because of its cardiac action. Noradrenaline ('levophed') in saline may be given by intravenous drip at a rate of 4 to 8 mcg./minute, the rate of infusion being regulated according to the blood pressure response. Whilst some desperately shocked patients may be rescued by such therapy, survivors have a poor further outlook, and, in general, severely shocked patients suffer a 75 per cent. or higher mortality.

The logical way to prevent *recurrence or spread of infarction* is to give a quick-acting anticoagulant.

For the *ventricular arrhythmias*, quinidine is the drug of choice. Quinidine sulphate, 3 to 5 grains (0.2 to 0.3 g.) four times a day, will damp out extrasystolic irregularities and will control paroxysmal ventricular tachycardia. For the latter, if established and threatening failure, the drug may be given by intravenous drip (0.3 g. in 500 ml.) run at 40 drops a minute under ECG control, or procainamide ('pronestyl') may be given intravenously: slowly in divided doses of 100 to 200 mg. up to a total of 1 g. In my experience, such measures have often proved efficacious and apparently life-saving (fig. 6).

As for the later complications which may lead to a fatal outcome, *left ventricular failure* and *congestive cardiac failure* are treated along standard lines. For the former, aminophylline (0.48 g. intravenously), with morphine (15 to 20 mg. [$\frac{1}{4}$ to $\frac{1}{3}$ grain] hypodermically), is highly effective. For congestive failure, digitalis, mersalyl and salt and fluid restrictions are required. Digitalis should be given if failure threatens, or if atrial fibrillation develops with a high ventricular rate, the risk attendant on its use (increased ventricular excitability) being less in such cases than the danger of its omission. *Cardiac rupture* cannot be prevented, but its incidence is reduced by a strict regime of rest and nursing care during the four to six weeks which are required for replacement of autolysed softened muscle by firm scar tissue.

The *embolic risks* may be systemic, from detachment of mural clots in the left ventricle, or pulmonary, from similar clots in the right ventricle or more often from thrombosed leg veins later in convalescence. The incidence of such thrombo-embolic incidents is reduced by about half by efficient

anticoagulant therapy, and the incidence of fatal attacks is cut even more dramatically (Tulloch and Gilchrist, 1950).

ANTICOAGULANT THERAPY

Opinion is still sharply divided as to the efficacy of anticoagulants in the treatment of myocardial infarction, and eminent cardiologists here and abroad are to be found in each of the opposing camps. In my opinion, anticoagulant drugs should neither be dismissed as 'rat poison' on the one hand, nor hailed as an elixir vitæ on the other. They have a place in treatment, and large series of patients treated with anticoagulants show a mortality half of that in comparable series without such treatment (e.g. Gilchrist and Tulloch, 1956). Despite this great reduction in mortality, however, there remain many instances in which they fail to prevent a fatal issue. Naturally, congestive failure, the dangerous arrhythmias and such disasters as cardiac rupture are not influenced by their use, but repeatedly one has seen instances of clinical recurrence of infarction in patients under treatment and in whom the prothrombin time had been adequately prolonged. Despite these disappointments, however, I would not willingly be without their aid.

There is general agreement as to the reduction they achieve in thromboembolic incidents and fatalities but less agreement regarding their effects in limiting spread or preventing recurrence of myocardial injury. From the nature of things, unless heparin is given by the general practitioner when he first sees the patient, if administration is delayed till the patient is in hospital on the second or later day of illness, such delayed therapy can have no effect on the early formidable mortality referred to above. There are those who follow Rushek and Zohman (1954), dividing patients into 'good risk' and 'bad risk' categories on clinical grounds (e.g. severity, degree of shock, persistence of pain) and who claim that for the 'good risk' cases anticoagulants have little to offer. I regard this as a dangerous fallacy, for it is common experience to see a patient following a mild attack, not shocked, a 'good risk' case in all respects, who a few hours or days later develops a full-blown major infarction and enters the 'bad risk' group. I know of no way of predicting which initially good risk case will suffer this change, and prefer to give anticoagulants to both groups.

It is probably unjustified to give this treatment to very old and frail subjects (75 and over), or to patients already severely handicapped after two or three earlier infarcts who develop yet another. Because of the risk of grave hæmorrhage, anticoagulants are contraindicated in patients with a history of peptic ulcer, so also they are dangerous after cerebral vascular accidents, and in patients with advanced liver disease.

All patients having anticoagulant drugs must be under close observation for hæmorrhage (skin, urine, stools, sputum), and the drug action must be checked by laboratory blood tests (coagulation time for heparin, prothrombin time for the coumarol group). When anticoagulants are used,

the appropriate antidotes must be available to cut short their action if desired (e.g. with hæmorrhage). These are protamine intravenously for heparin, and vitamin K by injection (or vitamin K1 by mouth) for the coumarol group.

In practice, if anticoagulant treatment is decided upon the use of rapid-acting (heparin) and slow-acting (coumarol group) drugs is combined for the first two days. By the third day the action of the coumarol drug is fully developed and heparin is discontinued. A standard regime is to give heparin 10,000 units (100 mg.) intravenously four- or six-hourly, for the first two days, together with an initial loading dose of a coumarol drug. Dicoumarol itself I regard as too dangerous, from its prolonged and delayed action. Phenindione ('dindevan'), 150 to 200 mg. on the first day and 100 mg. on the second (maintenance dosage thereafter 50 to 100 mg. daily), and ethyl biscoumacetate ('tromexan') in higher dosage, 600 mg. on the first day, 300 mg. on the second day (maintenance dose 300 to 600 mg. daily) are both satisfactory. The former is less persistent in action and cheaper than the second.

The maintenance daily dose of either drug is highly individual for each patient, and must be found by repeated laboratory estimation of the so-called 'prothrombin time'. The aim should be to maintain this time in seconds at about 2½ times that of control (normal) blood. Some pathologists report the results as a 'prothrombin index', which should be kept around 25 to 30 per cent. of normal by varying the dose of anticoagulant. Levels under 20 per cent. are needlessly low and at 10 per cent. there is risk of gross bleeding. Conversely, levels over 40 per cent. are therapeutically ineffective.

A full therapeutic level should be maintained for at least four weeks, since the risk of phlebothrombosis persists until the patient is ambulant.

DURATION OF REST IN BED

It is my practice to recommend six weeks' rest in bed for a case of average or severe type, particularly when the ECG indicates through-and-through (transmural) infarction. Shorter terms (two to four weeks) are permissible for patients with clinical and electrocardiographic evidence of minor infarction. Each patient passes through stages of increasing liberty, with more pillows, feeding himself, washing, and finally sitting up on the edge of the bed, before actually rising. Whilst complete rest is desirable for all, some patients must be allowed to rise to a night-stool for toilet, since their attempts to master the use of a bedpan impose far greater physical strain. This is particularly true of elderly and obese patients.

CONVALESCENCE AND AFTER-CARE

In general, a patient should be off work and convalescing at home for as long as he has been in hospital: i.e. six weeks for an average case, making in all three months off work. Great clinical judgment and tact on the part of the practitioner are needed, since some vigorous men are intolerant of

this delay, whilst others are timorous, over-cautious, and tend to become permanent semi-invalids. A great deal of cardiac neurosis results from the whole affair—the pain of the attack, the rush to hospital, the strict regime of rest and the knowledge, now widespread, that this is a commonly fatal illness. Further, the placing of the handicapped survivor in suitable employment may prove a difficult task, but one in which the doctor's active interest and help are vital.

Long-term anticoagulants have been under trial in an attempt to prevent recurrent episodes of infarction, particularly in younger men or in those who have had more than one attack in a few months. The value of the method has not yet been finally assessed. The prothrombin time in patients on such treatment must be checked weekly. For patients in country districts, it is feasible to send the blood by post to the laboratory, provided it arrives within twenty-four hours (Watson, 1956; Neilson and Briggs, 1957).

SUMMARY

(1) Attention is drawn to the common occurrence of cases without pain, or with pain of atypical distribution or mimicking a digestive upset, in which misdiagnosis is all too easy.

(2) A single normal electrocardiogram is not to be taken as excluding infarction, or even as rendering the diagnosis unlikely, if the clinical picture is characteristic.

(3) Of patients surviving long enough to be seen by a doctor, some 25 to 30 per cent. die in the first six weeks, and of these, half die in the first week and one-third within the first twenty-four hours.

(4) Treatment with anticoagulants cuts the over-all death rate by half, reduction of late thrombo-embolic deaths being partly or chiefly responsible. The risks of congestive failure and of dangerous arrhythmias are naturally unaltered, as is the formidable early mortality which occurs before anticoagulant treatment is begun.

(5) Whilst the general pattern of the risks to be run is known, risks diminishing as the days and weeks pass, there is sufficient unpredictable hazard to the individual patient to preclude the wise doctor from oversanguine assurance to the relatives, whatever be his optimism to the patient.

(6) The late prognosis is equally unpredictable for the individual, although with each year that passes the chances of prolonged survival increase.

(7) Many survivors become understandably apprehensive and introspective and need much help and reassurance.

(8) A healed myocardial infarct is compatible with many years of active life and work.

References

Gilchrist, A. R., and Tulloch, J. A. (1956): *Scot. med. J.*, **1**, 1.
Neilson, D. B., and Briggs, E. M. G. (1957): *Ibid.*, **2**, 284.
Rushek, H. I., and Zohman, B. L. (1954): *Lancet*, **ii**, 910.
Tulloch, J. A., and Gilchrist, A. R. (1950): *Brit. med. J.*, **ii**, 965.
Watson, H. (1956): *Lancet*, **i**, 541.

THE EPIDEMIOLOGY OF CORONARY DISEASE

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The New Epidemiology of the chronic non-infectious diseases has three aims: to sight the emergence of fresh threats to the public health, to look for clues about their origin in the mass behaviour of the specific disease and to test the consistency of theories of causation derived from experiments or observation in other fields of medical research. The rising menace of ischæmic heart disease is a matter of ordinary clinical experience and epidemiological evidence of varying quality is increasingly cited in support of views on its character and cause. An outline of the sources and nature of this epidemiological evidence may thus be useful.

National vital statistics, as always, provide the basis of much argument on the reality of the reported rise in cardiac mortality as well as its causation: and, too often, the inherent vagaries of the material are forgotten. Even within the same country time trends in disease death rates are bedevilled by changes in nomenclature and the official classification and coding of the complex syndromes of cardiovascular-renal failure leading to death. Changing fashions in diagnostic labelling may be deceptive; at least some of the rise in the death rate from coronary thrombosis could be accounted for by a switch from other forms of cardiovascular disease. International comparisons are even more treacherous; the contrast of cardiac mortality among British and Bantu assumes an equality of medical care and accuracy of birth and death registration which is unrealistic. Proportional rates based upon hospital admissions take no account of the mass of infectious illness in primitive communities which forms the bulk of admissions and allow fewer people to reach the age of circulatory disease.

Despite these hazards in interpretation, certain vital statistical facts resist an easy cynical explanation. In this country, for example, the ratio of male to female death rates at ages 45-54 from angina and coronary artery disease has increased sharply since 1910. Yet there is no reason to suppose that the family practitioner has different diagnostic standards for patients of different sex. The death rate from coronary disease in males of 50-54 in the United States is, at 450 per 1000, nearly double the corresponding rate for England and Wales of 229. The Canadian values of 378 per 1000 are intermediate. Again, the male:female ratios for the age-group 50-54 vary from 1.4 in Japan to 4.4 in Finland. Thus, there are major disparities in coronary-

disease experience between different times and different places which may be due to some real divergence in habits or environment. Of the many educated guesses about the elements involved, some have come from further epidemiological studies. In others, epidemiological surveys form the touchstone of their likelihood.

PSYCHOLOGICAL STRESSES IN POPULATION GROUPS

Ever since men had hearts with which to fear, the psychological stresses of life have been accused of causing heart disease, and the impressions of clinician and patient alike make this view entirely credible. Ryle and Russell (1949), reviewing the steep rise in coronary disease mortality in the last thirty years, thought that the concentration of the disease in the males of the professional classes suggested the effect of the quickening tempo of modern life on those with most responsibility to bear. The implications of the high ratio of coronary disease among physicians and surgeons in 1930-32 to that of all males of the same age (368 per cent.) are, on this hypothesis, peculiarly gratifying to the profession; the notion that this excess was due to the 'strain of medical practice' had its financial repercussions in the Spens award. Arnott (1954) has taken a less charitable view: 'The life of a physician in Birmingham is surely much less stressful than that of a peasant in the Yangste valley, with the ever-present menace of flood, famine, pestilence and war'. The high rates of coronary disease in the middle-aged American male are certainly consistent with the mental picture of the thrusting dynamic executive whose inner conflicts form the theme of so many films. Hollywood, however, holds a distorting mirror up to life, and psychological theories will not explain all aspects of the distribution of coronary disease mortality in America or anywhere else.

Although the disparity between the standardized mortality ratio for angina pectoris of the city clerk (148) and the agricultural labourer (31) may be due to the drift of the more ambitious striving personalities to town jobs with their associated anxieties and tensions, periods of presumptive stress, as during the occupation of Norway and the Netherlands, have not been remarkable for any general increase in cardiac death rates. On the other hand, certain observations on heart disease in general can be more readily explained by psychological than by other factors.

An investigation of the mortality experience of members of the German Bundestag in the years after the war, when political tensions were severe, showed that the deaths among these parliamentarians during the first session were significantly above expectations based on current national experience, whilst the rate among members of the provincial legislatures was rather fewer than anticipated (Boehm, 1953). In Canada, Buck (1955) has shown that patients with functional psychoses have a raised death rate from cardiovascular disease. Coming nearer home, it is difficult to suggest satisfying physical reasons for the post-1948 excess in coronary disease mortality

among general practitioners over the level in specialists in other branches of the profession (Morris *et al.*, 1952).

There is therefore no very good evidence that psychological stresses in large populations can account for long-term trends in the death rate from coronary disease; but there are hints that acute anxieties in exposed groups may precipitate death from coronary disease. Any such effect, however, is not specific to ischæmic forms of circulatory failure.

ALCOHOL, TOBACCO AND CORONARY THROMBOSIS

Much of the discussion about the origin of cardiovascular disease centres round the experience of specific occupational groups. Circulatory disorders, with coronary thrombosis as their severest manifestation, have been categorized as 'manager-krankheit' by German writers who have looked for causes in the working lives of the managerial group in industry and administration. As they point out, however, the attainment of personal eminence may be only another expression of the driving and restless personality which leads men into positions of responsibility and power, and exposes them to the incidental hazards to cardiac health which go with a generous expenses account: too much coffee, alcohol, overeating and inactivity. Before the occupational concentration in the managerial class is put down to the mental stresses of their job, these other factors have to be taken into account.

Cerebrovascular lesions	207
Myocardial diseases	161
Angina pectoris	128
Nephritis	233

TABLE I.—Circulatory disease mortality and access to alcohol. Standardized mortality ratio (1930-32) for inn and hotel keepers. Age adjusted (35-64): national male ratio=100. (Registrar General's Decennial Supplement 1931, Pt. IIa).

The effects of alcohol on the cardiovascular system can be deduced from the mortality rates among barmen and hotel keepers or in countries like France where consumption is high. Compared with all men of the same age, workers in occupations with easy access to alcohol have relatively high death rates from circulatory disease in general (table I). The same results appear in France where, in the wine-growing areas, high death rates from alcoholism and cirrhotic liver disease are associated with high mortality from cerebrovascular accidents, nephritis and, to a lesser degree, with diseases of the heart (Ledermann, 1953). As in psychological stresses, the presumptive effects of alcohol consumption are apparently not confined to coronary disease. The emphasis in both occupational and geographical associations between consumption and disease is on cerebral hæmorrhage and chronic nephritis. There is comparatively little epidemiological evidence about the effects of caffeine.

Indulgence in tobacco has been much more intensively studied by epidemiological methods in the context of lung cancer, and some of the best evidence for the influence of smoking on coronary disease mortality comes

as incidental findings in two major prospective surveys: Hammond and Horn (1954); Doll and Hill (1956). In both, smoking habits by type and amount of tobacco smoked were elicited by questionnaire from large groups of middle-aged men and the subsequent mortality experience of these men was noted by follow-up. Although the gradient was steeper in the American results, summarized in table II, both studies agreed in finding, particularly

Amount smoked per day (packs)	Age-group			
	50-54	55-59	60-64	65-69
0	349	689	886	2,084
$<\frac{1}{2}$	589	769	1,345	2,171
$\frac{1}{2}-$	741	1,284	1,950	2,712
1+	856	1,477	1,772	2,329

TABLE II.—Smoking habits and coronary disease death rates in the United States of America (Hammond and Horn, 1954).

in the younger age-groups, a definite association between the amount of tobacco smoked as cigarettes and the risk of dying from coronary thrombosis. This confirms the results of earlier controlled retrospective inquiries (English *et al.*, 1940) which showed, again among the younger men, a more frequent history of heavy smoking given by the patients with coronary disease. Cigarette smoking is thus clearly associated with ischæmic heart disease; but this need not necessarily imply that the relation is one of cause and effect. It is conceivable that both habit and disease are different expressions of some inner discontent. If, on the other hand, anxiety and stress are, as already suggested, at a fairly stable level in any community over a period of time, the corresponding rise in cigarette consumption is consistent with the increasing frequency of this particular form of circulatory failure. There is, too, a significant correlation between cigarette consumption (but not total tobacco consumption) and ischæmic disease mortality in the same year in the twelve countries for which records are available (de Fonseka, 1957). Although the fall in tobacco consumption at the end of the 1939-45 War has not resulted in any change in the tide of coronary mortality, sufficient epidemiological evidence is accumulating to suggest that cigarette smoking plays some part in precipitating ischæmic heart disease among younger men.

PHYSICAL ACTIVITY AS A MODIFYING INFLUENCE

Table III contrasts the standardized mortality ratios for occupations with the highest and lowest death rates in the period, 1930-32. As Stocks (1951) and Rentoul (1951) have pointed out, these disparities between professional and manual workers could be just as readily explained by the differing physical demands of the jobs as by the mental stresses involved in them.

Morris *et al.* (1953) have followed this up by showing that, in contrast to angina, the attack rate from serious infarction is much higher amongst actively employed bus conductors and postmen than in men working in sedentary jobs as drivers or clerks. There was, too, an indication of the better prognosis after infarction in the actively employed which may account for the gradient in mortality among men of the same social class

Highest		Lowest	
Physicians and surgeons	368	Sawyers and wood turners	50
Proprietors of wholesale businesses	235	Coalminers below ground making and repairing roads	49
Judges, barristers, solicitors	227	Coalminers below ground conveying material to shaft	41
Clergymen of Anglican Church	218	Plumbers	40
Ministers of religion other than Anglican or Roman Catholic	211	Coalminers below ground engaged in other work	40
Employers and managers	190	Stone miners and quarriers	38
Bank and insurance officials	183	Agricultural and gardeners' labourers	32
		Workers in chemical processes	20

TABLE III.—Mortality from angina pectoris and coronary disease by occupation. Standardized mortality ratios (20-64) in 1930-32. (Registrar General's Decennial Supplement, 1931, Pt. I).

when their jobs are independently ranked according to the physical demands they make. Some of these differences, however, may equally well arise from selective recruitment and wastage to other jobs. Only a fit man could contemplate a lifetime running up and down the stairs of a double-decker bus and Morris (1956) has shown, by an ingenious anthropometric survey based on uniform sizing, that the recruits to driving do differ in their physique from conductors.

DIET AND THROMBO-EMBOLIC PHENOMENA

Of all the circumstances of life which have been put forward to explain the distribution and natural history of coronary disease, dietetic indiscretion has excited the most urgent debate. In the past few years, there has been an accumulation of evidence which is, to say the least, suggestive. From the pathologist comes the observation that the atheromatous lesions thought to be the antecedents of thrombosis and infarction contain cholesterol; and lesions which mimic these in man can be produced by feeding rabbits on diets rich in that substance. Experiments on animals and men have given no completely satisfying assessment of the likely effect of diet on the production of ischæmic heart disease and most of the evidence comes from vital statistics and field surveys.

During the 1939-45 War, for example, there was a sharp fall in the Norwegian death rate from ischæmic heart disease which coincided with a reduction in the consumption of animal fats. In the hospitals, there was

a decreased incidence of thrombo-embolic phenomena (Strøm, 1951). Coronary disease death rates among middle-aged males are low in countries such as Japan (Keys, 1956) where much fish is eaten, or Italy where olive oil provides much of the dietary fat. In more primitive communities, maize and soya bean are prime articles of diet and the incidence of ischæmic heart disease is reputedly low among them. Within this country, there is a parallel

Social class	Males		Married women	
	1930-32	1950	1930-32	1950
I	237	150	157	92
II	148	110	126	93
III	95	104	93	101
IV	66	79	85	100
V	67	89	88	108

TABLE IV.—Social class trends in arteriosclerotic (coronary) heart disease. Standardized mortality ratios for ages 20-64. (Registrar General's Decennial Supplement, 1951, Occupational Mortality. Pt. I).

between the male social class gradient of coronary disease mortality and the proportion of the diet made up by animal fats.

There are clearly good reasons for suspecting that highly saturated animal fats in the Western dietary may be as dangerous as they are delectable. But before going back to a diet of porridge and skimmed milk, some inconsistencies in the evidence should be recognized. Whilst the reported fall in coronary disease mortality in the occupied countries certainly coincided with a fall in the animal fat content of the national diet, it also coincided with a fall in the supply of cigarettes. The same cycle of events in Denmark during the rationing of the 1914-18 War was strikingly associated with the consumption of spirits during the same period (Hindhede, 1922). Further, as Yerushalmy and Hilleboe (1957) have recently pointed out, there are many exceptions to the general rule that a high proportion of fat in the national diet is associated with a high mortality from ischæmic heart disease. Indeed, from a technical point of view the correlation between coronary mortality and total calories per person in the country concerned is higher than the corresponding association with the percentage of fats. In fact, the highest correlation is between heart disease ratio and the difference between energy requirements and available calories per head in the national populations. Nor will changes in the fat content of the diet explain either all the upsurge in coronary disease in the last thirty years, or the curious reversal in female mortality with social classes between 1931 and 1951 (table IV). This is not to say that dietary fat is not important; it simply means that dietary habit patterns will not explain all the observed facts of the distribution of ischæmic heart disease mortality.

A SUMMING UP

From his observations on the fat of thrombi in the walls of the coronary vessels, Duguid (1955) inferred that thrombosis rather than atheroma was the essential lesion in infarction leading to death; and this view agrees with much of the epidemiological evidence. Morris (1951), for example, has shown that the increased incidence of cardiac infarction and rupture found at necropsy in the London Hospital was not accompanied by any increase in the frequency of atheroma and calcification. The relationships found between quite varied factors and coronary disease mortality suggest that there is no single explanation of all the observed facts about the natural history of ischæmic heart disease. It seems rather that we are dealing with a disease, or a group of diseases, whose evolution amongst individuals predisposed by sex or constitution is furthered by the cumulative effects of different factors altering the blood-clotting mechanism. Some, like high-fat meals, may accelerate clotting whilst others, like tobacco, may accentuate its effect by adding vascular spasm. Exercise, on the other hand, may modify the functional results of vascular damage.

The epidemiological evidence is still not strong enough to incriminate any particular agent as the major determinant of coronary disease. The interim verdict must therefore be the canny Scots one of 'not proven'.

References

Arnott, W. M. (1954): *Brit. med. J.*, **ii**, 891.
Boehm, C. (1953): 'Hundert Jahre Victoria-Versicherung, 1853-1953. Die Zusammenstellung besorgte Wolfgang Knochenhauer'. Berlin.
Buck, C. (1955): *Canad. Serv. med. J.*, **11**, 791.
de Fonseka, T. E. J. (1957): Personal communication.
Doll, R., and Hill, A. B. (1956): *Brit. med. J.*, **ii**, 1071.
Duguid, J. B. (1955): *The Practitioner*, **175**, 241.
English, J., *et al.* (1940): *J. Amer. med. Ass.*, **115**, 1327.
Hammond, E. C., and Horn, D. (1954): *Ibid.*, **155**, 1316.
Hindhede, M. (1922): *Brit. med. J.*, **ii**, 248.
Keys, A. (1956): *Ibid.*, **ii**, 98.
Ledermann, S. (1953): *Concours méd.*, no. 16-19.
Morris, J. N. (1951): *Lancet*, **i**, 1, 69.
—— (1956): *Ibid.*, **ii**, 569.
——, *et al.* (1952) *Brit. med. J.*, **1** 503.
——, ——, (1953): *Lancet*, **ii**, 1053, 1111.
Rentoul, E. (1951): *Ibid.*, **i**, 1587.
Ryle, J., and Russell, W. T. (1949): *Brit. Heart J.*, **11**, 4, 370.
Stocks, P. (1951): *Lancet*, **i**, 351.
Strøm, A., *et al.* (1951): *Ibid.*, **i**, 126.
Yerushalmy, J., and Hilleboe, H. E. (1957): *N.Y. St. J. Med.*, **57**, 2343.

DIETARY FAT AND CORONARY HEART DISEASE

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THE possible role of diet, and in particular of the quantity and quality of dietary fat, in the etiology of coronary thrombosis, and perhaps more generally of atherosclerosis and its complications, has aroused intense interest and considerable controversy. The flood of publications during the last three or four years has been reviewed rather fully in two recent reports: a symposium on 'Fats in human nutrition' sponsored by the Council on Foods and Nutrition of the American Medical Association (Council on Foods and Nutrition, 1957), and a report to the American Heart Association and the American Society for the Study of Arteriosclerosis, published in full in *Circulation* (Stare *et al.*, 1957a), and more briefly in the *Journal of the American Medical Association* (Page *et al.*, 1957).

In this article I shall attempt only to summarize what advice can reasonably be given by a medical practitioner to a patient who is at risk of coronary thrombosis, and who has heard that dietary modification might have a favourable effect on his life and health expectation. I am one of those who believe that a case has been made for dietary modification on the part of those who are at risk; that is, those who have already had a coronary thrombosis, those who suffer from effort angina, or those who have a strong family history of coronary heart disease. Even though the case is not scientifically proven, the patient is entitled to make his own experiment, provided the dietary advice given by the doctor does not conflict with established principles of nutrition education.

At the same time, the opinion will be expressed that, whereas there is a strong case for an open mind and further research, the situation is too fluid and uncertain to justify pronouncement by those who administer laws about food production and who control nutrition education. In the past, dietetics has been a rich field for quackery, and public health administrators are wise to avoid far-reaching recommendations until the case for action is secure. A spate of premature and therefore undesirable lay publicity on the relationship between dietary fat and coronary heart disease evidently prompted the report to the American Heart Association, which in turn in the lay press has been misinterpreted as pouring cold water on recent research (*New York Times*, July 16, 1957). The authors of that report, however, while stating that 'the evidence at present does not convey any specific implications for drastic dietary changes', nevertheless make the reservation that 'these conclusions obviously apply to the general population, and not to the patients or individuals with a strong family history of early deaths from cardiovascular disease, who are being observed with some

regularity by their physician. Here, the newer concepts of nutrition readily suggest various types of diet therapy that may prove useful to certain patients'. Whilst not in agreement with some of the views expressed by the authors of the American Heart Association report, I find myself in full agreement with these general conclusions.

The case for dietary modification on the part of those who are specially at risk of coronary thrombosis will be stated in the form of four postulates and a conclusion. The postulates will then be reviewed and the practical recommendations arising out of the conclusion will be elaborated.

FOUR POSTULATES

(1) In the last two or three decades there has been a very disturbing rise in mortality and severe morbidity from acute coronary thrombosis and/or occlusion (the term 'coronary occlusive episodes' will be used; the word 'episode' implying an acute clinical event) and resultant myocardial infarction.

(2) The variation in the reported incidence of coronary heart disease in different population groups at different times in these last two or three decades suggests that at least the acute occlusive episodes can be favourably or unfavourably influenced by easily modifiable environmental factors.

(3) The mean level of certain lipid or lipoprotein compounds in the blood, and particularly of total serum cholesterol, is statistically associated in various population groups with the incidence of reported coronary occlusive episodes in such a way as to suggest causality in the association.

(4) The level of serum cholesterol can be influenced upwards or downwards by dietary modification, and particularly by changes in the quantity and quality of the dietary fat.

Tentative conclusion from postulates.—If postulates (1) to (4) are accepted, it appears logical to conclude that the incidence of coronary occlusive episodes in people at risk could be reduced by modifications of diet, particularly if they lead to sustained reduction of the serum cholesterol. This tentative conclusion does not imply that unsuitable diets are the only, or even the most important, cause of coronary heart disease; it implies only that dietary modification can improve life expectation in people at risk. The conclusion will depend for its validity upon the reliability of the four postulates, which will now be critically examined.

POSTULATE 1

In 1955, an editorial in the *Lancet* (1955), when examining this postulate, stated that 'all cardiologists whose experience goes back thirty years or more seem to agree with the vital statisticians that the higher mortality rates reflect a real increase in coronary artery disease, and also that young people below the age of forty, are now affected much more often than formerly'. Jolliffe (1957) quotes this editorial and comes to the same conclusion. Nevertheless, a responsible group of investigators (Stare *et al.*, 1957a) has questioned the validity of the statistical evidence. Such striking differences

of opinion probably indicate differences of terminology or definition. The latter group define their aim as being 'to evaluate evidence for and against the concept that the fat content of the average present-day North-American or North-European diet is a significant factor in the genesis of cerebral, myocardial, renal or peripheral atherosclerosis'. They rightly conclude that 'to date there is no incontrovertible evidence for such a relationship'. In their conclusion, however, they discuss myocardial infarction as though coronary atherosclerosis were the only factor in its causation. This in spite of the fact that a small section of the report entitled 'fat and clotting' implicitly recognizes the possible importance of the short-term events leading up to occlusive episodes.

It is therefore necessary to define some terms. The term coronary heart disease is commonly used to include coronary artery disease and its effects. These are chiefly ischæmic and include angina pectoris, coronary insufficiency, coronary thrombosis and/or occlusive episodes, myocardial infarction and fibrosis and their late sequelæ. Although the term, coronary artery disease, should include such conditions as thromboangiitis obliterans, other rare obstructive conditions and even syphilitic occlusion of the coronary ostia, it is often used as synonymous with coronary atheroma and atherosclerosis which is probably the cause of 95 per cent. of coronary heart disease. The whole problem of atheroma and atherosclerosis as they affect the coronary arteries has been discussed by Morgan (1956) under the heading *coronary occlusion*. The latter may occur insidiously but commonly presents itself as an acute clinical coronary episode. The episode may be due to intravascular coagulation of blood sufficient to occlude the remaining lumen in a segment of coronary artery previously rendered narrow or irregular by the process of atheroma or atherosclerosis. Alternatively, it may be due to rupture of an atheromatous plaque, or some other mechanism whereby the balance between the remaining lumen of a coronary artery, its collateral channels and the oxygen need of the myocardium is suddenly upset. These mechanisms are not at present understood and terminology is confused (Oliver and Boyd, 1957). It is evident, however, that clear distinction must be made between atherogenesis as a slowly developing process and the short-term events which lead to the occlusive episode.

Evidence has been advanced by Morris (1951) that in the London population the incidence of coronary thrombosis has increased markedly with very little if any change in the extent or severity of coronary atherosclerosis in the same population. If this information is correct, the rising mortality from coronary heart disease must be attributed to factors which precipitate occlusive episodes. There is nothing inherently improbable in this implication; in fact, it would reasonably explain the decline in mortality from coronary thrombosis in Scandinavian countries during the last few years of the 1939-45 War and its fairly rapid return to original rates in the first few years after the war (Biorck, 1956). It is unlikely that there would be a substantial change during so short a period of time in two directions in the

severity or extent of atherosclerosis in the Scandinavian population. The effects of altered dietary and other environmental influences could much more probably be related to the incidence of coronary occlusive episodes through the mechanisms of thrombogenesis than through the mechanism of atherogenesis when periods less than a decade are under consideration.

The evidence derived from life assurance statistics has recently been reviewed and the cautious conclusion is reached that 'it is difficult to evaluate the real trend of mortality from coronary disease' (Lew, 1957). This author, however, notes the large sums of money being expended by the Life Insurance Medical Research Fund in investigation of this field.

The practitioner with a patient at risk is therefore justified in accepting the first postulate as a reasonable basis for action.

POSTULATE 2

Fluctuations in mortality from coronary thrombosis or coronary artery disease in the Scandinavian countries during and immediately after the second world war have already been referred to (Biorck, 1956). Differences in the incidence of coronary heart disease in different races throughout the world and in different socio-economic groups within a given population have often been reviewed (Keys, 1956, 1957). In brief, it can be stated that there appear to be striking differences between many privileged European nations and many underprivileged nations. Many of the latter are pigmented people and it is not impossible or inherently improbable that genetic differences might be responsible through the mediation, for example, of different types of lipid metabolism. On the other hand, it is difficult to avoid the conclusion that in some parts of the world there is a positive correlation, within a genetically homogenous population, between mortality from coronary artery disease on the one hand and occupation and social and economic stratification on the other. In general, social and economic underprivilege and occupations involving physical work appear to protect, whereas social and economic overprivilege and sedentary occupations tend to render population groups more susceptible.

There are undoubtedly possible fallacies in this reasoning.

For example, the observation by Morris *et al.* (1953) that London bus-drivers have a higher incidence of coronary thrombosis than London bus-conductors was at first interpreted as evidence of an adverse effect of either sedentary occupation or tension and strain. Later, Morris himself produced evidence that the difference might be genetic in that the stature of bus drivers was generally considerably greater than that of bus-conductors (Morris *et al.*, 1956).

This gap in the argument which might, for example, apply to the differences reported in Britain's five socio-economic groups (Registrar General, 1954) cannot at present be completely closed. It could not, however, upset the conclusion that the temporal fluctuations in the Scandinavian mortality figures are due to modifiable environmental influences.

Among the modifiable influences there are theoretically many but only a few can be supported by serious evidence. Those most canvassed are diet (Keys, 1953), physical activity (Mann *et al.*, 1955), tension and strain

(Friedman and Rosenman, 1957) and perhaps smoking (Hammond and Horn, 1954; Doll and Hill, 1956). To review the evidence in support of the non-dietary influences would be irrelevant in this study. None of them is inherently improbable but there is no need to choose between them and diet. It may possibly be that all, and even others, are operative. The case for diet is that it is easily modifiable and that the evidence in support of it is quite considerable—at least as much as in the case of any other. The second postulate is also consistent with the report of the WHO 'Study group on atherosclerosis and ischæmic heart disease' (1957), especially the section on 'Relation of diet to production of thrombosis'. It is therefore held to be reasonable.

POSTULATE 3

Provided the first two postulates are accepted the evidence for a close statistical association between the mean level of total serum cholesterol in middle-aged men and mortality from coronary occlusive episodes is almost overwhelming. There can be no doubt of the differences in mean serum-cholesterol level in males at a given age between different racial and population groups (Bronte-Stewart *et al.*, 1955; Keys, 1956, 1957). There can be no question that these differences correlate with differences of reported morbidity and mortality in respect of coronary occlusive episodes in the same population and racial groups (Vogelpoel and Schrire, 1955; Keys, 1957). No clinician would be surprised to learn that there was a close statistical correlation between serum-cholesterol levels and incidence of coronary heart disease. The relationship between coronary heart disease, serum-cholesterol level and diseases such as diabetes mellitus, myxœdema and familial hypercholesterolæmia would prepare him for such an association.

With regard to other fractions of the lipids and lipoprotein complexes of the blood I propose to adopt the view that, whatever the operative fraction or fractions may be in the causation of coronary thrombosis, the changes can be conveniently measured by simply determining the total serum cholesterol which acts as an index. This view has recently been accepted by all but one of a responsible panel of investigators (Gofman *et al.*, 1956).

POSTULATE 4

Before discussing this postulate a word must be said about spontaneous fluctuations in the level of serum cholesterol. These are quite considerable under ordinary conditions of living.

> For instance, among ten subjects studied weekly for twelve weeks while living their ordinary life of daily activity and without modifying their ordinary diet the variation about the mean serum-cholesterol level for the group was plus or minus 20 mg. per cent., and the biggest and lowest fluctuations about the individual means were plus or minus 30 mg. per cent. and plus or minus 14 mg. per cent. respectively (Gordon and Brock, 1957).

This means that under ambulant conditions single readings of the serum cholesterol must not be given very great weight in relation to the healthy mean for that age or sex. It means further that experiments on dietary modification of serum cholesterol must include many consecutive readings

both in the experimental and control periods. Under conditions of hospitalization, and particularly under the conditions of a metabolism ward, fluctuations in the serum cholesterol from day to day or from week to week under standard conditions are considerably smaller, and significant differences can be obtained from five consecutive readings in a ten-day period. Allowance must always be made in interpretation, however, for uncontrollable variations resulting from minor digestive disturbances or periods of relative failure of appetite or of diarrhœa.

With these reservations, there can now be no reasonable doubt that the serum cholesterol can be significantly affected by changes in the diet, and particularly by changes in the quantity and quality of the dietary fat. This subject has been exhaustively reviewed by Bronte-Stewart *et al.*, 1956; Ahrens *et al.*, 1957; Malmros and Wigand, 1957.

Many differences distinguish 'Westernized' diets from the diets of underprivileged populations in whom the incidence of coronary heart disease is low. Most of these have been reviewed elsewhere (Brock and Bronte-Stewart, 1955) with the conclusion that none is more important in relation to coronary heart disease than the fat content. It is not denied that others may be contributory. The evidence for the role of quantity versus quality of dietary fat has been reviewed by Brock and Gordon (1957). It is concluded that the theory that serum cholesterol responds simply to the fat/calorie ratio of the diet is no longer acceptable; quite a number of fats, particularly the unsaturated oils, actually lower the serum cholesterol when substituted isocalorically either for carbohydrate or for the saturated fats. They even have the power to neutralize the cholesterol-raising effect of the saturated fats when they are added, with consequent increase in calories, as well as when they are isocalorically substituted.

The evidence for this view has recently been critically reviewed by Jolliffe (1957). He described the differences between saturated and unsaturated fats succinctly in the following words:—

'One of the most important chemical properties of fat is that pertaining to the double bonds in the fatty acid chains. The unsaturated fatty acids, such as oleic, linoleic, linolenic, and arachidonic have multiple double bonds, while the saturated fatty acids such as stearic and palmitic have no double bonds. Fats are mixtures of these fatty acids. Those with a high proportion of unsaturated fatty acids, because of these double bonds, are more active chemically. It is for this reason that they easily become rancid, readily pick up extraneous tastes and odours, and have high iodine numbers. On the other hand, fats containing mostly saturated fatty acids are chemically less active. They store longer without rancidity, do not readily pick up extraneous tastes and odours, and have low iodine numbers. In addition, fats that are highly saturated are plastic at ordinary temperatures while those highly unsaturated are liquid'.

In general, the animal fats are saturated whilst vegetable and marine oils are unsaturated. There are, however, important exceptions to this general rule: coconut oil, for example, has an iodine number or value of 8 to 10, which makes it more saturated than the animal fats. The chain length of a fat has also to be taken into account in considering its biological value, although little is yet known on this score. Even the biological effects of the

unsaponifiable residues have been canvassed (Beveridge *et al.*, 1957).

Much interest has been taken in the effect of commercial hydrogenation of unsaturated oils.

By this process, double or unsaturated bonds are saturated artificially in order to alter the texture, melting point and other qualities of easily available vegetable oils. The process is extensively used in the manufacture of margarine or cooking fats. The work of several groups has shown that hydrogenation of an unsaturated oil may alter its biological effect on the serum cholesterol (Bronte-Stewart *et al.*, 1956; Ahrens *et al.*, 1957; Malmros and Wigand, 1957).

There is obviously an inference that commercial hydrogenation of oils might be contributing to the rising incidence of coronary heart disease. It would be most unwise and unfair to industry to come to premature conclusions on this point. In the first place there are many processes of hydrogenation and they may not all have the same biological effect in relation to a given change in iodine value or melting point.

For instance, the biological value appears to be altered by the formation of geometric isomers even though the isomers retain their double bonds and hence their iodine numbers (Privett *et al.*, 1955; Sreenivasan and Brown, 1956). Secondly, many margarines and cooking fats consist of mixtures of hydrogenated and natural oils and, on the principle of neutralization referred to above, the latter may neutralize any theoretical detriment which may have occurred in the former through the process of hydrogenation.

Conjecture in this field has run ahead of evidence since Sinclair (1956) published his highly interesting and thought-provoking but speculative review. All that can safely be said at present is that different fats have different effects on the serum-cholesterol level. There are many possible reasons for these differences, but the saturation-unsaturation theory gives a reasonable basis for action.

The mechanism of the raising and lowering effect of different oils and fats on the serum-cholesterol level has been investigated in part. It has been suggested that fats are transported in the blood as cholesterol esters, the solubility of which depends upon the degree of saturation of their fatty acid component, and that those with a saturated fatty acid component deposit more readily on or in the intima of the blood vessels (Schroeder, 1956). A different approach is the capacity of the unsaturated fats to promote the excretion of cholesterol from the body (Ahrens *et al.*, 1957). The theory that cholesterol is excreted as bile acids has substantial experimental backing (Gordon *et al.*, 1957a, b; Lewis, 1957) and is a reasonable theory.

THE TENTATIVE CONCLUSION

If postulates (1) to (4) are accepted, then the tentative conclusion as stated is reasonable in so far as it applies to people at risk of coronary thrombosis. It implies that in respect of such persons reduction of serum-cholesterol levels in excess of the mean for age and sex is desirable if this can be achieved by simple dietary modification, and that such reduction might improve the expectation of life and health in the person at risk. It implies further that the required modification can be reasonably easily made by adjusting the quan-

tity or quality of fat in the diet without adversely affecting the individual's health in other respects. These implications will now be further examined.

It is reasonable to suppose that there is a healthy range of serum cholesterol and that, even if the latter is wide and genetically determined, there must be possible disadvantages attached to hypercholesterolæmia. The analogy with blood pressure is close: hypocholesterolæmia and hypercholesterolæmia are as difficult to define as hypotension and hypertension; both must be correlated with age and sex; in both, the normal range for age and sex is wide and is at least partly genetically determined. Neither must the mistake be made of categorizing as abnormal extremes within the normal range which are determined by genetic predisposition and which may be healthy for that individual. The analogy with hypertension goes further; at some point normal tension merges into hypertension which is definitely adverse in its effects. Pathological hypertension appears to result from an abnormal acceleration of normal age trends which then creates a vicious circle. There is no present evidence of a vicious circle mechanism in rising serum-cholesterol levels, but they would appear to be an abnormal acceleration of normal age trends. Fortunately, it has already been shown that simple changes in the diet are preventive in the majority of individuals.

The term 'middle-aged hypercholesterolæmia' is used here to distinguish a widely prevalent condition from the less common condition which we call 'essential hypercholesterolæmia'. The former is thought to be easily preventable; the latter is due to a strongly determined genetic trend, although even here there is some evidence that the trend can be modified by adjusting the diet (Malmros and Wigand, 1955; Ahrens *et al.*, 1957; Gordon and Brock, 1957). It has been suggested (Aldersberg, 1951) that essential hypercholesterolæmia is the homozygous expression of a gene which in its heterozygous form produces middle-aged hypercholesterolæmia. In practice, however, middle-aged hypercholesterolæmia can be prevented to a considerable extent by reducing the quantity or altering the quality of dietary fat. The mechanism by which such a preventive dietary modification might be effective has been discussed under postulate 4. Its desirability is inherent in acceptance of postulate 3.

APPLICATION

It remains simply to demonstrate that the diets which reduce middle-aged hypercholesterolæmia can be made healthy and not unreasonably irksome. We have shown (Gordon and Brock, 1957) that this can be achieved without any dietary modification over periods of three months or more by the consumption daily of 50 to 75 grammes (four to six tablespoons) of sunflower or other highly unsaturated oils. Simple fat restriction by itself is not sufficient to lower the serum cholesterol unless the restriction is severe (Hatch *et al.*, 1955). But if 50 grammes of highly unsaturated oil are substituted for, say, 75 grammes of saturated fat in a diet containing 40 to 45 per cent. of calories from fat, the combined quantitative and qualitative change should be effective in reducing the serum cholesterol while leaving

the diet reasonably palatable. This has been our experience and that of Malmros and Wigand (1957).

As the chain of reasoning through the four postulates to the tentative conclusion and application is not as yet conclusive, I am opposed to the application of the implied conclusions to modification of nutrition education or food regulations. On the other hand, I feel that the reasoning is so suggestive that it justifies application to modification of the diets of those who are at risk of coronary thrombosis and who have either middle-aged or essential hypercholesterolæmia. How are the implications of the tentative conclusion to be applied in practice?

'Weight reduction' and 'fat restriction' must be clearly distinguished (Brock and Gordon, 1957). There can be no question at all of the importance of weight reduction in patients who are fat, obese or markedly overweight. Fatness or obesity must be clinically estimated and is not the same as overweight.

In view of what has been said about spontaneous fluctuations in the level of blood cholesterol no reliance should be placed on a single reading of the serum cholesterol, even from an experienced laboratory. The case for dietary modification must be judged in each case in relation to the trend of blood cholesterol over several readings, and the estimate of the extent to which the patient is at risk of coronary occlusion. If possible, patients should not be made 'blood cholesterol conscious'.

Restriction of total fat in the diet should not be pressed too far. A reasonable objective is 30 per cent. of calories from fat (National Nutrition Council of South Africa, 1956). At this level weight can be maintained and the palatability of the diet is not seriously impaired. As knowledge advances, it may be possible to allow higher intakes of fat if the distribution of the fats is moved considerably over from the saturated to the unsaturated varieties. Knowledge on this score is still insufficient for definite recommendation. There is no need to reduce cholesterol intake as such since there is no experimental or theoretical justification for believing that the cholesterol content of commonly used 'Western' diets contributes materially to the level of serum cholesterol (Keys *et al.*, 1956).

PRACTICAL DIETETICS

For intelligent patients who have reasonable restraint and self-discipline it is sufficient to give the following general recommendations. Cream should be avoided and butter should be used sparingly. Full-cream milk should be restricted to half a pint (300 ml.) a day for an adult, or skimmed milk should be used. Visible fat should be cut off and rejected from meat as served on the plate. Stews prepared from fat meat should be avoided. One of the highly unsaturated oils, such as sunflower oil, maize (corn) oil, or fish oil, should be used for fries, roasts, salad dressings and mayonnaise. Prepared dishes to which cream, butter or other fats and oils are added in the preparation, e.g. pastries and desserts, should be avoided unless the added oil

is highly unsaturated. Many physicians ban or limit eggs severely because of their cholesterol content. This is not a valid reason. On the other hand, there is experimental evidence to suggest that eggs raise the serum cholesterol out of proportion either to their fat or their cholesterol content. Until this point has been settled restriction of eggs is probably wise.

For patients whose temperaments require detailed diet sheets, or whose hypercholesterolæmia is severe, reference may be made to the diets published by Stare *et al.* (1957b).

FUTURE DIRECTIONS

It should be clear from what has been said that I am not overlooking the role of inheritance and sex or of other environmental factors such as exercise, tension and strain, smoking and hypertension. Nor do I need to be persuaded of the importance of multiple etiology in this group of diseases.

With respect to dietary management the future discovery of the reason for the favourable effect of highly unsaturated oils may lead to much simpler forms of treatment. Work is in progress in the search for active small-dose fractions of these oils. Preparations are already on the market; some of these are sheer quackery; others are based on as yet incomplete evidence. The present dietary recommendations are therefore tentative and may well be improved upon in the near future. For this reason I prefer not to go into detailed menus.

The question of the relationship of dietary fat to the etiology of slowly progressive atheroma and atherosclerosis is, of course, highly interesting and relevant. It is by no means impossible that the marked differences in coronary and aortic atheroma which have been described, for example, in Japanese and Bantu compared with Europeans *might* be due to genetic differences. The evidence, however, is at least suggestive that even in these races changing environment and particularly changing dietary patterns may increase the tendency to early and severe atheroma and atherosclerosis (Keys, 1957). Decision on this subject must await further research and evidence.

SUMMARY

(1) Recent work is reviewed on the relationship between dietary fat, serum cholesterol and coronary heart disease.

(2) Some ambiguous terms which have led to confusion are defined.

(3) A clear distinction must be drawn between the mechanisms of slowly progressive atheroma and atherosclerosis and the short-term mechanisms which lead to occlusive coronary episodes; the latter are primarily under consideration in the recommendations which follow.

(4) Four postulates are submitted which appear to be supported by a considerable body of evidence.

(5) From these postulates the conclusion is drawn that it appears logical at present to conclude that the incidence of occlusive coronary episodes in people at risk could be reduced by modifications of diet, particularly reduction in the quantity or alteration in the quality of fat.

(6) Suggestions are made as to the advice which can reasonably be given by a practitioner to a patient who is at risk of coronary heart disease and who has heard that dietary modification may favourably influence his prognosis.

(7) It is concluded that the evidence is not yet decisive enough to justify altered general advice on food patterns and nutrition education for apparently healthy people, or for the prevention of atherosclerosis.

(8) The views expressed must be taken into reasonable relation with non-dietary environmental causes and admitted genetic and sex factors.

References

Ahrens, E. H., *et al.* (1957): *Lancet*, **i,** 943.
Aldersberg, D. (1951): *Amer. J. Med.*, **11,** 600.
Beveridge, J. M. R., Connell, W. F., and Mayer, G. A. (1957): *Fed. Proc.*, **16,** 11.
Biorck, G. (1956): 'World Trends in Cardiology', Vol. 1, edited by A. Keys and P. D. White, New York, chap. 3.
Brock, J. F., and Bronte-Stewart, B. (1955): *Minn. Med.*, **38,** 852.
——, and Gordon, H. (1957): *S. Afr. med. J.*, **31,** 663.
Bronte-Stewart, B., Antonis, A., Eales, L., and Brock, J. F. (1956): *Lancet*, **i,** 523.
——, Keys, A., and Brock, J. F. (1955): *Ibid.*, **ii,** 1103.
Council on Foods and Nutrition (1957): *J. Amer. med. Ass.*, **164,** 1890.
Doll, R., and Hill, A. B. (1956): *Brit. med. J.*, **ii,** 1071.
Friedman, M., and Rosenman, R. H. (1957): *Circulation*, **16,** 339.
Gofman, J. W., *et al.* (1956): *Ibid.*, **14,** 691.
Gordon, H., and Brock, J. F. (1957): (In the press; see also Gordon *et al.*, 1957a).
——, Lewis, B., Eales, L., and Brock, J. F. (1957a): *S. Afr. med. J.*, **31,** 594.
——, ——, ——, —— (1957b): *Lancet*, **ii,** 1299.
Hammond, E. C., and Horn, D. (1954): *J. Amer. med. Ass.*, **115,** 1316.
Hatch, F. T., Abell, L. L., and Kendall, F. E. (1955): *Amer. J. Med.* **19,** 48.
Jolliffe, N. (1957): *N.Y. St. J. Med.*, **57,** 2684.
Keys, A. (1953): *J. Mt Sinai Hosp.*, **20,** 118.
—— (1956): *J. chron. Dis.*, **4,** 364.
—— (1957): *J. Amer. med. Ass.*, **164,** 1912.
—— *et al.* (1956): *J. Nutr.*, **59,** 39.
Lancet (1955): **ii,** 1123.
Lew, E. A. (1957): *J. chron. Dis.*, **6,** 192.
Lewis, B. (1957): *S. Afr. J. Lab. clin. Med.*, **3,** 316.
Malmros, H., and Wigand, G. (1955): *Minn. Med.*, **38,** 864.
——, —— (1957): *Lancet*, **ii,** 1.
Mann, G. V., *et al.* (1955): *New Engl. J. Med.*, **253,** 249.
Morgan, A. D. (1956): 'The pathogenesis of coronary occlusion', London.
Morris, J. N. (1951): *Lancet*, **i,** 1, 69.
——, *et al.* (1953): *Ibid.*, **ii,** 1053, 1111.
——, ——, (1956): *Ibid.*, **ii,** 569.
National Nutrition Council (1956): *S. Afr. med. J.*, **30,** 108.
Oliver, M. F., and Boyd, G. S. (1957): *Brit. Heart J.*, **19,** 582.
Page, I., *et al.* (1957): *J. Amer. med. Ass.*, **164,** 2048.
Privett, O. S., Pusch, F. J., and Holman, R. T. (1955): *Arch. Biochem.*, **57,** 156.
Registrar General (1954): 'Decennial Supplement England and Wales, 1951, Occupational Mortality', Part 1, p. 13, H.M. Stationery Office, London.
Schroeder, H. A. (1956): *Lancet*, **i,** 1017.
Sinclair, H. (1956): *Ibid.*, **i,** 381.
Sreenivasan, B., and Brown, J. B. (1956): *J. Amer. Oil Chem. Soc.*, **33,** 341.
Stare, F. J., *et al.* (1957a): *Circulation*, **16,** 163.
——, —— (1957b): *J. Amer. med. Ass.*, **164,** 1920.
Vogelpoel, L., and Schrire, V. (1955): *Lancet*, **ii,** 1108.
World Health Organization (1957): 'Study Group on Atherosclerosis and Ischæmic Heart Disease', WHO Technical Report Series No. 117, Geneva.

HORMONES AND CORONARY DISEASE

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During the earlier part of this century the classical clinical features of most common endocrine diseases and the clinical syndromes of coronary atherosclerosis both became widely recognized. Great progress has been made in the understanding of endocrine diseases and of coronary disease but it is not generally appreciated that there is any relationship between them. In this review some associations between hormones and coronary disease will be described, but first it is necessary to consider briefly the disturbances of the circulating lipids so often found in patients with clinical coronary disease. The term 'clinical coronary disease' is used to denote the clinical syndromes of coronary atherosclerosis—angina and myocardial infarction.

THE CIRCULATING LIPIDS AND CORONARY DISEASE

Cholesterol was first demonstrated in atherosclerotic plaques by Vogel, in 1847, and soon afterwards Virchow (1856) suggested that atheromatous lesions were probably due to primary imbibition from the passing blood resulting in fatty metamorphosis in the arterial intima. Since Anitschkow and Chalatow (1913) observed atherosclerotic plaques in rabbits fed cholesterol in vegetable oil, intimal lesions have been produced in many species by various experimental regimes. In general, the extent and number of cholesterol-induced experimental lesions are proportional to the degree of hypercholesterolæmia, although this is not to say that hypercholesterolæmia is a *sine qua non* for the development of typical atheromatous lesions. In man, however, the lipid composition of atherosclerotic plaques and of the plasma is similar enough to suggest that the lipids of the plaques have their origin in the plasma, and labelled cholesterol studies have shown that in man this plasma lipid can be incorporated into atherosclerotic plaques. Since most of the cholesterol in the human arterial wall is probably derived from the plasma, it is important to determine the character of the circulating lipids in patients with atherosclerosis.

There is little doubt that the circulating lipids are disturbed in many patients with clinical coronary disease, and this disturbance has been demonstrated by the measurement of several dependent variables. The plasma cholesterol, the plasma cholesterol/phospholipid ratio, the β-lipoprotein cholesterol (the cholesterol carried by the β-globulins) and some low density lipoprotein molecules are all elevated in many patients with clinical coronary disease (Gertler *et al.*, 1950; Barr *et al.*, 1951; Jones *et al.*, 1951; Oliver and Boyd, 1953b, 1955b). These abnormalities are most frequent and marked in young patients with clinical coronary disease, and the etiology of the disease may be more closely related to a disorder of lipid metabolism in

young patients than in older patients. These disturbances of the circulating lipids have been demonstrated in apparently healthy men before the development of coronary disease and are not merely associated with the onset of angina or myocardial infarction.

There are several possible explanations for this association of abnormalities of the circulating lipids with clinical coronary disease. First, the association

Age	Numbers of cases		Ratio of men to women
	Men	Women	
Younger than 35	19	1	19:1
35-39	44	3	15:1
40-49	188	26	7:1
50-59	256	53	5:1
60-69	195	86	2:1
Older than 70	68	61	1:1

TABLE I.—The sex and age distribution of 1000 consecutive cases of clinical coronary disease attending Edinburgh Royal Infirmary.

might be due to chance but statistics make this possibility highly improbable. Secondly, hypercholesterolæmia might lead to the development of intimal arterial lesions. Thirdly, it is possible to argue that the hypercholesterolæmia results from the presence of such lesions. Fourthly, both the hypercholesterolæmia and the intimal lesions could stem from some common cause. Whatever the explanation this association occurs sufficiently often to be regarded as an integral part of the etiology, and the influence of hormones on the circulating lipids has therefore an etiological and potentially therapeutic interest.

HORMONAL INFLUENCES ON THE CIRCULATING LIPIDS

PHYSIOLOGICAL

Menstrual cycle.—Depression of the plasma cholesterol occurs regularly at ovulation. The endogenous secretion of œstrogens also undergoes regular cyclical variation and is generally believed to be maximal immediately before ovulation. Although most of the assay methods for the hormones which integrate to yield the repetitive pattern of the menstrual cycle are only capable of quantitative or semiquantitative evaluation, œstrogens have been assayed with greater precision than others, and it is therefore permissible to correlate the cyclical changes in the circulating cholesterol with œstrogen secretion. It has been suggested that depression of certain plasma lipids at ovulation could result from the increased endogenous secretion of œstrogens at that time (Oliver and Boyd, 1953a).

Pregnancy.—Elevation of the plasma cholesterol occurs during the last trimester of normal pregnancy. This hypercholesterolæmia has been variously ascribed to the influence of the fœtus, to the metabolic demands of lactation, to placental toxins and to increased adrenal synthesis of cholesterol, but the evidence to implicate these factors is circumstantial and unconvincing and it is more likely that endogenous hormonal secretions contribute

to the hypercholesterolæmia (Boyd, 1934; Oliver and Boyd, 1955a). In pregnancy, however, there is increased œstrogenic, thyroid and adrenocortical activity and, since the administration of œstrogens, thyroxine and cortisone all lower the plasma cholesterol in humans, the development of hypercholesterolæmia is not yet satisfactorily explained on a hormonal basis.

Associated disease or disorder	Number of patients
Endocrine disorders:	
Diabetes mellitus	3
Myxœdema	3
Spontaneous premature menopause	2
Bilateral ovariectomy	2
Cushing's disease	1
Hypertension	5
During pregnancy or puerperium	4
Anæmia	1
No associated disease	4

TABLE II.—An analysis of the health of 25 women who developed clinical coronary disease before the age of 40.

Menopause.—Elevation of the plasma cholesterol has been observed during the first post-menopausal decade in healthy women, and might be related to the decrease in œstrogenic activity and to any gradual post-menopausal decrease in thyroid and adrenocortical activity. The incidence of clinical coronary disease is low before the menopause and increases rapidly thereafter (table I). In women who develop clinical coronary disease before the age of 40, coexisting endocrine diseases are common (table II).

PATHOLOGICAL

Diabetes mellitus.—The levels of the circulating cholesterol and the circulating glucose are only weakly correlated, but in general there is hypercholesterolæmia in association with uncontrolled glycosuria and hyperglycæmia. Since many diabetic patients have occult or overt vascular disease, it is unlikely that a comprehensive study of the circulating lipids will be made in diabetic patients in whom vascular disease can be excluded. Even although hypercholesterolæmia is common in young diabetics, it is difficult to determine whether the hypercholesterolæmia is due to the diabetes *per se* or whether it is associated with coexisting vascular disease or renal damage. Thus, although hypercholesterolæmia is often present in diabetes, it has not been clearly established that it is due to insulin lack.

The occurrence of angina in a diabetic patient was first described by Seegen in 1864, and subsequently it has been shown that coronary atherosclerosis and clinical coronary disease are common in diabetic patients (Joslin, 1930). Provided they have the disease long enough, diabetics will almost invariably develop atherosclerosis, and of 484 necropsies on patients who had had diabetes for five or more years only four were free from atherosclerosis (Warren, 1938).

Myxœdema.—It is well known that in myxœdema the basal metabolic rate

is lowered and there is hypercholesterolæmia (Mason *et al.*, 1930). There does not seem to be a simple inverse relationship between the basal metabolic rate and the circulating lipids, however, since dinitro-*o*-cresol can elevate the basal metabolic rate without reducing the hypercholesterolæmia of myxœdema, and conversely certain analogues of thyroxine reduce hypercholesterolæmia without apparently elevating the basal metabolic rate (Lerman and Pitt-Rivers, 1955).

Although myxœdema has its greatest incidence at the ages when atherosclerosis is most common, there have been frequent reports of an unusually high incidence of coronary atherosclerosis in myxœdematous patients (Black and Kampmeier, 1934; Bruger and Rosenkrantz, 1942). The observation of marked and widespread atherosclerosis in young patients with endemic cretinism and thyroid deficiency offers further support for a positive correlation between the two conditions.

Hyperadrenocorticism.—Hypercholesterolæmia and atherosclerosis have been observed in Cushing's disease but the significance of this association is uncertain as both could be explained by accompanying diabetes, renal damage, hypertension and depression of thyroid function.

Castration.—Hypercholesterolæmia has been observed in patients who have had a bilateral ovariectomy and in patients following orchidectomy.

Examination of the hearts of 49 women, in whom bilateral ovariectomy had been performed between the ages of 20 and 42 years, revealed more severe coronary atherosclerosis, decade by decade, than that observed in a comparable control group (Wuest *et al.*, 1953). No comparable necropsy observations have been made in male castrates.

EXPERIMENTAL

There have been numerous investigations into the influence of various hormones on the circulating lipids in many different species. Although this experimental approach has been very profitable, it should be emphasized that the action of a certain hormone in one species does not necessarily give any indication of its effect on the circulating lipids in another species.

In man the influence of administered hormones on the circulating lipids has recently been reviewed (Oliver and Boyd, 1956b) and can be summarized as follows. Corticotrophin, hydrocortisone and cortisone lower hypercholesterolæmia; thyroxine and its analogues lower hypercholesterolæmia; œstrogens, when given at dose levels sufficient to induce feminizing effects, also lower hypercholesterolæmia. Insulin lowers the hypercholesterolæmia associated with diabetes but probably has no action on the circulating lipids in non-diabetic subjects. The administration of androgens results in hypercholesterolæmia. Growth hormone, glucagon and progesterone have not been shown to produce any significant alteration in the levels of the circulating lipids.

HORMONAL INFLUENCES ON THE ARTERIAL WALL

Atherosclerosis is a generalized disease and commonly involves the abdominal

aorta more extensively and earlier than any other vessel. Usually, the presence of intimal plaques has little significant effect on the function of the large lumen of the aorta, whereas comparable thickening in the smaller coronary and cerebral arteries may cause partial or total occlusion of the lumen with severe impairment of its function. Although the incidence of coronary atherosclerosis is not much less than that of the aorta, the consequences of coronary atherosclerosis are more disastrous than aortic atherosclerosis.

An agent which increases the tonicity of arteries by producing contraction of the muscular elements in the arterial wall reduces the vascular bed and necessitates a compensatory increase in dynamic pressure. Any such rise in intravascular hydrostatic pressure favours the development of atheromatous lesions, and this is exemplified by the observation of a positive correlation between hypertension and clinical coronary disease, particularly in women (Master, 1953). Prolonged vasoconstriction results in compression of the vasa vasorum by the contracting media, produces ischæmia in the arterial wall and can hinder the normal diffusion of tissue fluids. Hormones can influence the arterial wall by altering vasomotor tone and capillary resistance. For example, certain adrenal steroids increase vasomotor tone whereas thyroxine and œstrogens cause peripheral vasodilatation. Adrenaline and noradrenaline cause vasoconstriction and elevation of the blood pressure in many vascular beds although the coronary arteries are usually dilated. An increase in capillary resistance occurs after various forms of stress or after the administration of corticotrophin and insulin. Capillary permeability is decreased by corticotrophin and cortisone.

HORMONAL INFLUENCES ON BLOOD COAGULATION

Blood coagulation can be regarded as consisting of two opposing groups of dynamic processes: those which favour the formation of thrombin and fibrin, such as the factors which contribute to the formation of thromboplastin; and those which prevail against the formation of thrombin and fibrin, such as heparin, antithromboplastin and fibrinolysis. Both these groups of process can be influenced *in vivo* by hormones. For example, the administration of corticotrophin and cortisone during the treatment of collagen diseases was followed by shortening of the clotting time and an increase in the incidence of thrombo-embolic incidents; moreover, corticotrophin and cortisone inhibit fibrinolytic activity. Adrenaline and experimentally induced stress exert diverse effects on the coagulation system, including acceleration of the clotting time and an increase in fibrinolytic activity. In some animals thyroid-stimulating hormone, deoxycortone acetate and androgens increase fibrinolysis whilst growth hormone shortens clotting time. These observations refer only to whole blood clotting time and fibrinolysis, but evidence is accumulating that hormones can influence several of the phases of coagulation.

HORMONAL IMBALANCE AS AN ETIOLOGICAL FACTOR IN CORONARY DISEASE

It has been shown that the physiological and pathological secretion and the experimental administration of hormones can influence the circulating lipids, and in certain endocrine diseases there is an increased incidence of clinical coronary disease. In addition, hormones can influence the coronary arterial wall, chiefly by altering vasomotor tone. There is also evidence that hormones can influence the coagulation system. It is possible therefore that the maintenance of normal cholesterol metabolism, of normal vasomotor tone and of the coagulation-fibrinolysis system could all be disturbed by an alteration of the physiological endocrine balance. Such an endocrine imbalance might therefore contribute to the development of coronary atherosclerosis and of clinical coronary disease. This suggestion does not deny in any way the significance of dietary factors in the development of coronary disease, and evidence is accumulating that the quantity and character of dietary fat are important in the etiology of this disease. Even in countries where the fat consumption is high most of the population does not suffer from clinical coronary disease, and of five men, whose environment and responsibilities are apparently closely comparable, only one may be expected to develop it. There is, then, an individual susceptibility, which is not well understood and may be partly determined by heredity.

The nature and precision of each individual's endocrine balance may well be determined constitutionally by heredity, and some subjects are more susceptible than others to extraneous influences which tend to disturb this balance. Inability to compensate adequately for changes in environmental responsibilities might be transmitted to the endocrine system by way of the cerebral cortex and hypothalamus and result in disturbance of normal endocrine balance. Acute 'stress' can lead to profound endocrine disturbances. For example, the normal pituitary-adrenocortical balance can be upset and lead to manifestations such as hypoglycæmia, gastro-intestinal erosions, hæmoconcentration and eosinopenia. The normal pituitary-thyroid balance can be upset by acute 'stress', and the rapid development of thyrotoxicosis following a sudden severe shock or acute anxiety is common and well recognized. The normal pituitary-gonadal balance can also be upset by acute psychological duress; for instance, the sudden cessation of menstruation following emotional anxiety, and impotence of psychological origin have often been described. Thus, the relationship between neural stimuli and endocrine gland function is close and this subject has recently been reviewed by Harris (1955).

The principal weakness of the theory that an endocrine imbalance may play an etiological role in the production of coronary disease lies in the fact that no marked consistent abnormality of endocrine gland function has yet been found in patients who have the clinical features of this disease. It is unlikely that there is any gross endocrine disturbance in coronary atherogenesis, as this would have wide metabolic repercussions and would have

been recognized years ago, but it is possible that a comparatively minor disturbance may have passed unnoticed, due to the insensitivity of the available methods for assessing endocrine gland function. Indeed, the evidence at present obtained from standard tests of endocrine function is not adequate to confirm or refute the theory.

HORMONE THERAPY IN CORONARY DISEASE

Hypercholesterolæmia and coronary atheroma.—In the future, hormones may be used therapeutically to lower the hypercholesterolæmia associated with clinical coronary disease. It has been shown in the experimental animal that the degree and extent of cholesterol deposition in the arterial wall are directly proportional to the degree of hypercholesterolæmia and reduction of hypercholesterolæmia is followed by less cholesterol deposition. It is reasonable to argue therefore that reduction of the plasma cholesterol in man might result in the passage of less cholesterol from the arterial lumen through the arterial wall to the veins and lymphatics of the adventitia and, perhaps in less cholesterol deposition within the arterial wall.

At present the widespread metabolic actions of the adrenocortical hormones preclude their use as depressants of plasma cholesterol. Thyroxine and its analogues are generally unsuitable for administration to patients in whom the myocardial blood supply is impaired by coronary atherosclerosis, since any increase in the myocardial demands and oxygen consumption might be expected to result in ischæmia and aggravate symptoms. There is, however, some evidence that certain thyroxine analogues will lower hypercholesterolæmia without apparently elevating the basal metabolic rate (Lerman and Pitt-Rivers, 1955; Oliver and Boyd, 1957). Unfortunately, these analogues may also cause selective increase in myocardial oxygen consumption without influencing the gross metabolic rate. If it becomes possible to lower the circulating cholesterol without increasing the oxygen requirements of any tissue, these analogues of thyroxine will have potential therapeutic importance. Although œstrogens in suitable doses are reasonably tolerated by many men and achieve a 25 to 30 per cent. decrease in plasma cholesterol (Oliver and Boyd, 1956a), the feminizing effects are likely to limit their therapeutic value in the control of hypercholesterolæmia. Unfortunately, it has not been possible to counteract these feminizing effects with androgens without also antagonizing their cholesterol depressant action.

There is as yet no entirely satisfactory means for the continued control of the circulating lipids and there is no conclusive evidence that such control will retard the atherosclerotic process. The ultimate assessment of any regime designed to lower the plasma cholesterol must depend upon its effect on morbidity and mortality. Once clinical coronary disease has become manifest, inhibition of the atherosclerotic process (if it can be achieved) will not necessarily alter the prognosis.

Myocardial infarction.—In man, adrenaline causes an increase in cardiac output and a fall in peripheral resistance without much effect on the blood

pressure; in contrast, noradrenaline causes no change in the cardiac output, but increases peripheral resistance and produces a rise in blood pressure. Noradrenaline has been used extensively in the treatment of hypotension following sympathectomy and in the treatment of shock. It has also been used to combat the hypotension and peripheral circulatory collapse which can follow acute and severe myocardial infarction. Although most of the patients treated in this way had an initial pressor response, the use of noradrenaline for the shock of severe infarction has generally been disappointing. The pressor response is often difficult to maintain and serious arrhythmias occur in approximately 10 per cent. of patients. Some reports have been encouraging, but many of the recoveries have occurred in patients not very severely shocked. Noradrenaline has not been very impressive in really severely shocked cases, but it is possible that its early administration may be a life-saving measure in a proportion of patients. It must be given intravenously in a continuous saline infusion and this involves the administration of comparatively large amounts of fluid to a circulatory system whose efficiency is temporarily impaired; one result of this is that pulmonary œdema and congestive cardiac failure are not uncommon in the convalescent period.

Angina pectoris.—Atherosclerotic coronary arteries are often unable to supply sufficient blood for the normal myocardial demands, and myocardial ischæmia may result but, if the myocardial demands could be restricted, this reduced blood supply might be adequate. In the hope that the development of myocardial ischæmia and of anginal attacks might be lessened, various attempts have been made to reduce the myocardial oxygen requirements and thereby reduce the work of the heart in patients with clinical coronary disease. Thyroidectomy (Blumgart *et al.*, 1933), thiouracil (Raab, 1945) and radioactive iodine (Blumgart *et al.*, 1950, 1956) have all been used to reduce the rate of myocardial metabolism, and in a majority of patients a striking reduction of the incidence and severity of angina has followed.

Of these three methods of decreasing thyroid activity, it is probable that radioactive iodine is the most satisfactory for the treatment of angina. If possible, it is desirable to avoid anæsthesia and surgery in patients with advanced coronary disease. The thiourea derivatives do not always induce hypothyroidism in every patient. When effective they must be continued for the rest of the patient's life and at any time during such administration there is the danger of agranulocytosis and other drug reactions. If the initial dose of radioactive iodine is insufficient, additional doses can be given and, if the patient cannot tolerate the hypothyroid state, it can be relieved or abolished by the administration of thyroid. Patients who may be expected to gain the greatest benefit from radioactive iodine are those in whom intractable angina has been present for a year or longer. It is not advised for those patients who can be kept comfortable and moderately active by the use of nitroglycerin, and it is unwise to give radioactive iodine to those who are in the terminal stages of the disease, particularly since between two

and six months intervene before the maximum benefit can be expected.

There is some experimental evidence that androgens cause peripheral vasodilatation. With the object of improving the blood supply by increasing the collateral circulation or by inducing vasodilatation of the coronary arteries, testosterone propionate has been used in the treatment of angina. It has no specific effect on angina (Levine and Likoff, 1943), however, although some patients may experience a sense of well-being which allows the pain to be better tolerated.

SUMMARY

A positive relationship between hormones and coronary disease has been outlined.

The influence of hormones on the circulating lipids, on the arterial wall and on the coagulation system suggests that hormonal imbalance could contribute to the development of coronary disease.

At present hormones have no general therapeutic value in coronary disease, but they have been used in clinical research to correct hypercholesterolæmia, to combat the shock which may accompany acute myocardial infarction, and to relieve angina pectoris.

References

Anitschkow, N., and Chalatow, S. (1913): *Zbl. allg. Path. path. Anat.*, **24,** 1.
Barr, D. P., Russ, E. M., and Eder, H. A. (1951): *Amer. J. Med.*, **11,** 480.
Black, H. A., and Kampmeier, R. H. (1934): *Colorado Med.*, **31,** 262.
Blumgart, H. L., Freedberg, A. S., and Kurland, G. S. (1950): *Circulation*, **1,** 1105.
——, ——, —— (1956): *J. chron. Dis.*, **4,** 381.
——, Levine, S., and Berlin, D. D. (1933): *Arch. intern. Med.*, **51,** 866.
Boyd, E. M. (1934): *J. clin. Invest.*, **13,** 347.
Bruger, M., and Rosenkrantz, J. A. (1942): *J. clin. Endocr.*, **2,** 176.
Gertler, M. M., Garn, S. M., and Lerman, J. (1950): *Circulation*, **2,** 205.
Harris, G. W. (1955): 'Neural control of the pituitary gland', London.
Jones, H. B., *et al.* (1951): *Amer. J. Med.*, **11,** 358.
Joslin, E. P. (1930): *Ann. intern. Med.*, **4,** 54.
Lerman, J., and Pitt-Rivers, R. (1955): *J. clin. Endocr.*, **15,** 653.
Levine, S. A., and Likoff, W. B. (1943): *New Engl. J. Med.*, **229,** 770.
Mason, R. L., Hunt, H. M., and Hurxthарl, L. M. (1930): *Ibid.*, **203,** 1273.
Master, A. M. (1953): *Circulation*, **8,** 170.
Oliver, M. F., and Boyd, G. S. (1953a): *Clin. Sci.*, **12,** 217.
——, —— (1953b): *Brit. Heart J.*, **15,** 387.
——, —— (1955a): *Clin. Sci.*, **14,** 15.
——, —— (1955b): *Brit. Heart J.*, **17,** 299.
——, —— (1956a): *Circulation*, **13,** 82.
——, —— (1956b): *Lancet*, **ii,** 1273.
——, —— (1957): *Ibid.*, **i,** 124.
Raab, W. (1945): *J. Amer. med. Ass.*, **128,** 248.
Seegen, J. (1864): 'Der Diabetes Mellitus auf Grundlage Zahlreicher Beobachtungen dargestellt', Leipzig, p. 182.
Virchow, R. (1856): *Ges. Abh. z. wiss. Med.*, **14,** 458.
Vogel, J. (1847): 'The Pathological Anatomy of the Human Body', Philadelphia, pp. 386, 584.
Warren, S. (1938): 'The Pathology of Diabetes', 2nd edition, Philadelphia.
Wuest, J. H., Dry, T., and Edwards, J. E. (1953): *Circulation*, **7,** 801.

DIFFICULT EPISTAXIS AND ITS CONTROL

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It is fortunate that in the majority of cases, idiopathic nose-bleeding comes from an erosion of the wall of a small blood vessel on the front and lower part of the nasal septum in an 'area of predilection' first described by Little—an American surgeon—in 1879. It is frequent in children and young persons, is rarely of any serious significance, and because of accessibility is easy to control by finger pressure on the ala nasi, shrinking agents (adrenaline), astringents (tannic acid jelly), caustics (silver nitrate) or by charring with the actual cautery point.

The remaining cases comprise the small group in which the bleeding proves (in the words of the old writers) difficult, stubborn, obstinate, inveterate, copious, and sometimes alarming. This is more often seen in adult or elderly patients afflicted with arteriosclerosis, hyperpiesia, renal disease, and chronic venous thoracic congestion, and occasionally in apparently healthy people. Nasal bleeding can, of course, result from specific causes, for instance, in injury, fractures, new growths, blood dyscrasias, vitamin deficiencies, telangiectasia, but these will not be considered here.

THE VAGARIES OF EPISTAXIS

One difficulty in dealing with idiopathic nose-bleeding is that it is often capricious. It may start quietly or suddenly, flow profusely for a variable length of time, and then stop abruptly or gradually and by the time the surgeon arrives the episode is over and no obvious source can be found. There is a further annoying tendency for the bleeding to recur and continue intermittently for days, demoralizing the patient, relatives and doctor, and it may eventually lead to such a degree of exsanguination that blood transfusion may be required to avoid a fatal issue. In many cases it is true that the ultimate result is a spontaneous cessation of the bleeding, sometimes from sheer loss of blood, and within limits this is not always detrimental in patients with hyperpiesia or polycythæmia who may be relieved thereby from the more serious danger of intracerebral hæmorrhage. Mention of this consideration is sometimes advocated for the reassurance and encouragement of patients during expectant treatment, but few people in distress appear to derive consolation by the intimation of their deliverance from a greater evil.

At this stage expectant and simple treatment is well worth while. Trotter, Layton and many others have laid down the familiar rules which include the sitting posture, ice-cold water bathing of the face and neck, mouth-breathing and the suppression of swallowing which is apt to dislodge clot lying on the

upper surface of the soft palate. An injection of morphine, repeated if necessary after an hour or two, is valuable, as in hæmorrhage from other parts of the body. If the bleeding happens to originate in the accessible front portion of the nasal passage, plugging with cotton-wool or ribbon gauze soaked in 1 in 1000 adrenaline (with or without cocaine) may prove successful in controlling the bleeding at once but if, as so often happens, the blood flows from a point farther back then the plug is liable to increase the patient's discomfiture by directing an overflow into the naso-pharynx, throat and opposite nasal passage.

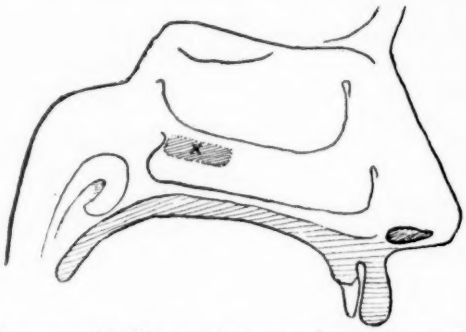

Fig. 1. — X = The usual source of posterior nasal hæmorrhage, hidden underneath the back part of the inferior turbinal.

SEVERE EPISTAXIS

Admission to hospital is, of course, essential for cases of severe epistaxis, but here another difficulty may arise because these emergencies tend to fall into the hands of junior and other members of the hospital staff who cannot be expected to have the confidence and skill needed to explore the posterior parts of the nose in the presence of profuse hæmorrhage. Moreover, it must be emphasized that in some cases a successful search has to be conducted by a trained rhinologist while the nose is actually bleeding. Mainly because of the obstacles of inaccessibility and limited equipment, the failure to find the bleeding point led the old writers and their textbooks to cover the lack of knowledge by falling back on a diagnosis of 'a general ooze from multiple points', and to depend largely upon expectant treatment. This omission has also resulted in the widely adopted and ineffective practice of general plugging of the nose and nasopharynx, and also in the use of various kinds of inflatable rubber balloons and other fanciful devices which in actual use often prove cumbersome and disappointing. Judging from recent literature, this failure to locate the bleeding spot is also responsible for the questionable and rather frequent resort to heroic surgical methods such as ligature of the carotid vessels in the neck, and an attack on the internal maxillary artery after a difficult approach through the posterior antrum wall—even then with unpredictable results.

THE SITE OF HÆMORRHAGE

In recent years, a method of control has been developed on simple surgical principles. It is now established that in most cases the eroded blood vessel lies in *the vault or lateral wall of the inferior meatus underneath the posterior one-third of the inferior turbinal* (fig. 1). This is where the descending palatine

vessels emerge from the spheno-maxillary fissure and start to diverge over the lateral nasal wall. Whether they are venous or arterial is of no practical moment, but in this area they lie superficially and without much support under the mucosa where they are prone to dilatation and rupture.

A number of writers have suspected the spheno-maxillary vessels from a general anatomical viewpoint, but hitherto only a few have focused attention convincingly on this precise region. It may be that a detailed cadaveric study of the blood vessels in this particular area would prove worth while and throw further light on the subject, but it is clear that the remote and awkward location is the reason why in the past the source of bleeding has been so difficult—not only to find, but also to control. Outside the line of vision, tucked up and protected underneath the rigid back end of the inferior turbinal, the blood sometimes gushes forth indefinably from the back of the nose like a mountain torrent, and even when subdued it manages to remain concealed by flowing gently over the upper surface of the soft palate and into the throat. Because of this protected locus an ordinary complete packing of the main nasal passage is often useless, even when reinforced by a post-nasal plug. There is no direct pressure on the bleeding point. But in the light of this new information, the most vicious hæmorrhage can be tackled with confidence. The surgeon can figuratively wade through the flow, plant the mop firmly on the hole, and then have time to look around.

TREATMENT

A technique of dealing with these cases is now fairly well established. After admission to hospital a prompt estimate of the degree of exsanguination, shock, blood pressure and kidney function is carried out and, if required, a blood transfusion is started. If the bleeding has stopped or is minimal the patient is propped up in bed on a back rest, given an appropriate injection of morphine, cleaned up and made comfortable, and sometimes this is all that is needed. If the bleeding persists or recurs, the patient is removed to the operating theatre and dealt with as a rhinological operation case, the following essential items of equipment being at hand:—A powerful illuminant (the preference is for an efficient focusing headlamp fitted with a 6 volt 0.3 amp bulb). A strong and dependable arrangement for spot suction through a fine brass tube of approximately $\frac{1}{8}$ inch (3 mm.) external diameter at its business end. An electro-cautery apparatus suitable for nasal work. A local anæsthetic and shrinking solution consisting of 20 per cent. cocaine hydrochloride in 1 in 1000 adrenaline solution, an eminently satisfactory combination which will not only open up the nasal passage and relieve the patient of the discomfort of the proceedings, but will also take the edge off the worst bleeding.

With the nose cleansed, packs removed, blood clots blown, sucked or lifted out with forceps, and the patient lying down, a rapid alternate application of suction and judicious painting with the local anæsthetic is conducted until the whole nasal passage is anæsthetized, cleansed and opened out for a

critical inspection. Any obvious and visible source of bleeding can now be immediately sealed with the cautery point, and no further treatment is required. If the bleeding appears to have stopped, as often happens during the preliminary preparation, and no obvious site is discovered, a search should be made underneath the posterior half of the inferior turbinal using a small dry wool mop on a fine carrier. As a rule it will be found that a repetition of this test will reveal a small but significant trickle of blood from this hidden spot.

Having confirmed the site, it remains to bring the leak under control by a specialized method of packing. Because of the danger of increasing the hæmorrhage the application of the cautery point is not recommended when the bleeding area is out of sight. If the nasal septum is much deflected from the midline and interferes with the manipulations there should be no hesitation in carrying out an immediate submucous resection so that the posterior part of the nasal passage can be adequately visualized, although there is no apparent virtue in this operation *per se* as a means of controlling epistaxis, and as recommended by a few surgeons.

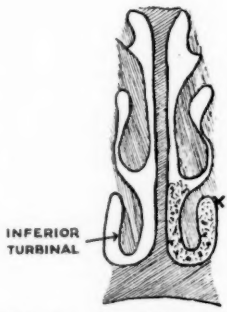

FIG. 2. — X = The gauze packing correctly placed for arresting hæmorrhage.

Using a fine septal elevator, the posterior half of the inferior turbinal is now gently infracted away from the outer nasal wall, and the corresponding part of the inferior meatus is filled with a paraffined 1-inch-wide (2.5 cm.) ribbon gauze pack, the first six inches (15 cm.) of which have been medicated with compound tincture of benzoin B.P. The turbinal is next replaced, and the remainder of the gauze firmly packed with downward pressure between the back part of the septum and the inferior turbinal, which brings full pressure to bear on the bleeding spot (fig 2). There is no need to pack the rest of the nasal passage or to distress the patient with a post-nasal plug. The benzoin tincture is antiseptic and induces a local inflammatory reaction which helps in the natural sealing of the eroded blood vessel. The ribbon gauze pack is gently removed on the 3rd or 4th day, and no further treatment is called for. Bleeding from other parts of the nasal passage can usually be controlled by standard methods.

SUMMARY

(1) In many cases difficult idiopathic posterior nasal hæmorrhage derives from an eroded blood vessel in the vault or lateral wall of the posterior third of the inferior meatus of the nose, under cover of the inferior turbinal.

(2) With average skill and proper facilities, and armed with this knowledge, the rhinologist should have little difficulty in mastering this problem.

(3) The frequency of involvement of this particular area warrants its classification as the second 'site of predeliction' for epistaxis.

GOWERS' MIXTURE

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THE original formula devised by Sir William Gowers for the well-known mixture used in the prevention of migraine was as follows:—

Sodii bromid.	5–10 grains
Liq. trinitrini	2 minims
Acid. hydrobrom. dil.	5 minims
Tinct. nucis vom.	5 minims
Tinct. gelsemii	5–10 minims
Syrup. limonis	60 minims
Aq.	ad ½ ounce

Ft. mist. sig. ½ ounce t.d.s., p.c.

Writing, in 1888, on the treatment of migraine, Gowers observes:

'In the majority of cases and especially in those in which there is conspicuous pallor during an attack, the drug that has the most influence is nitro-glycerine. Given regularly during the intervals, just as bromide is given for epilepsy, it has a striking effect in many patients, rendering the attacks far slighter and far less frequent and occasionally stopping them altogether. It should be given twice or three times a day, after food. If taken when the stomach is empty it passes rapidly into the blood and may cause brief cephalic discomfort which, though not objectionable in itself, sometimes deters the patient from continuing the medicine . . . By far the most convenient mode of prescribing it is in a 1 per cent. solution in alcohol which can be given in combination with tinctures or acids but is decomposed by alkalis. A very useful combination is with tincture of nux vomica, tincture of gelsemium and dilute phosphoric acid . . . I have found such combinations of the liquid preparation of nitro-glycerine with other drugs far more useful than the administration of nitro-glycerine tablets. It is not well to continue it during an attack; at the very onset a dose may be taken, but if this is not effective the medicine should be omitted till the attack is over; it seldom gives relief to symptoms and occasionally makes them worse. It is possible that the drug acts chiefly by periodically flushing the nerve centres with arterial blood and so improving the nutrition and function of the nerve cells'.

MODIFICATIONS

Various modifications of the original formula have been introduced. Kinnier Wilson's Neurology (Bruce, 1955) favours the following:—

Solution of glyceryl trinitrate	1 minim (0.06 ml.)
Sodium bromide	10 grains (0.6 g.)
Solution of strychnine hydrochloride	3 minims (0.2 ml.)
Orange-flower water	to ½ fluid ounce (14 ml.)

Walshe (1955) gives the following formula:—

Solution of glyceryl trinitrate	1 minim (0.06 ml.)
Solution of strychnine hydrochloride	3 minims (0.2 ml.)
Tincture of gelsemium	15 minims (1 ml.)
Phenazone	5 grains (0.3 g.)
Sodium bromide	5 grains (0.3 g.)
Chloroform water	to 1 fluid ounce (28 ml.) thrice daily

Brinton (1936) pointed out that all the different formulæ do, or should, contain nitroglycerin, around which the medicine was originally composed, and that it is necessary to prescribe this in an acid medium to avoid decomposition. He goes on to say that tincture of belladonna, 5 to 10 minims

(0.3 to 0.6 ml.) can also usefully be substituted for tincture of nux vomica.

It is of interest that in 'The Extra Pharmacopœia (Martindale)' 1952, three different prescriptions are given for Gowers' mixture. In the first (Gowers' original formula) the nitroglycerin is given in the form of the solution of glyceryl trinitrate. In the second (Pharmacopœia of Westminster Hospital) it is given as a $\frac{1}{200}$ grain (0.3 mg.) tablet of nitroglycerin taken at the same time as the medicine, and in the third (Pharmacopœia of Charing Cross Hospital) it is omitted altogether. The details of the second and third prescriptions are as follows:—

Westminster Hospital formula

Potassium bromide	10 grains (0.6 g.)
Tincture of gelsemium	10 minims (0.6 ml.)
Solution of strychnine hydrochloride	4 minims (0.25 ml.)
Dilute hydrochloric acid	2½ minims (0.15 ml.)
Chloroform emulsion	6 minims (0.4 ml.)
Water	to ½ fluid ounce

Nitroglycerin, $\frac{1}{600}$ grain (0.3 mg.) tablet, is given at the same time.

Charing Cross Hospital formula

Potassium bromide	10 grains (0.6 g.)
Tincture of gelsemium	10 minims (0.6 ml.)
Solution of strychnine hydrochloride	4 minims (0.25 ml.)
Dilute hydrochloric acid	5 minims (0.3 ml.)
Chloroform emulsion	10 minims (0.6 ml.)
Compound infusion of gentian	to 1 fluid ounce (28 ml.)

INCOMPATIBILITIES

Even though soluble phenobarbitone is now generally considered a more efficient sedative than bromide, it should not be prescribed as a substitute for bromide in Gowers' mixture as it is precipitated by the acid and is therefore incompatible. I was responsible for this error in my article on analgesics, in the symposium on 'Favourite Prescriptions' published in *The Practitioner*, last year (Clarke, 1957) and I am grateful to the chief pharmacist of St. George's Hospital for pointing the mistake out to me. He writes:

> 'While this precipitate is readily dispersible by shaking, it is considered bad pharmaceutical practice to dispense a mixture in this form and the pharmacist called upon to dispense it would almost certainly seek the permission of the prescriber to dispense the phenobarbitone separately, preferably in tablet form'.

CONCLUSION

Scientific medicine looks askance today at polypharmacy but there is every reason to believe that Gowers' clinical views were sound and it is for the pharmacologists to explain to us why this particular combination of drugs works so well.

References

Brinton, Denis (1936): *The Practitioner*, **136**, 520.
Bruce, A. Ninian (1955): 'Kinnier Wilson's Neurology', 2nd ed., London, vol. III, p. 1723.
Clarke, C. A. (1957): *The Practitioner*, **178**, 33.
Gowers, William (1888): 'Diseases of the Nervous System', vol. II, Edinburgh, p. 793.
Walshe, Francis (1955): 'Diseases of the Nervous System,' 8th ed., Edinburgh, p. 140.

CHARCOT'S JOINTS

A CASE RECORD

By GEORGE SPANOPOULOS, M.D.

Nicosia, Cyprus

CHARCOT's joints, once so common in clinical practice, can now be classified among the relative rarities of medicine. The following case is of interest in view of the dramatic onset.

CASE RECORD

History.—Mrs. A.B., aged 50, who had been married for 30 years and had no children, gave the following history. While standing at the sink one day, washing

FIG. 1.—Antero-posterior view showing complete disorganization of both knee joints, with marked new bone formation.

clothes, her legs suddenly gave way and she fell to the ground. She was unable to get up. At no time did she feel any pain. She had never before had any aches or pains in her legs and her only complaint was that she used to get easily tired. In describing her fall she compared it with the fall of a house of cards.

On examination the right knee joint was found to be dislocated and there was a

fracture of the right femur and the tibia. On the left side there was a fracture of the lower end of the femur. Both knee joints were completely disorganized. The Wassermann and Kahn examinations were not done as the patient refused permission.

X-ray examination confirmed the complete disorganization of both knee joints (fig. 1, 2). The right one was dislocated with fractures of the lateral condyle of the femur and the medial tuberosity of the tibia. On the left side there was a fracture of

FIG. 2.—Lateral view of knee joints.

the lower end of the femur. In both joints there was marked new bone formation, and on the left side 'lace-work' periostitis of the lower end of the femur.

The radiologist reported: 'the appearances are those of syphilis of the bone involving both knee joints (Charcot's joint of the hypertrophic type)'.

DISCUSSION

This case is of interest on account of the complete absence of symptoms before the sudden collapse of the knee joints, in spite of the extensive bony and joint damage revealed on radiological examination.

HEALTH INSURANCE SCHEMES IN NORTH AMERICA

By MICHAEL ARNOLD, L.R.C.P. & S.I.
Wembley, Middlesex

In the summer of 1957, I was fortunate enough to have the opportunity of paying a visit to North America. Of sun and good living there was plenty, but by far the greatest stimulant was gained from ferreting out my opposite number in general practice. It was refreshing and provocative of much discussion to discover the problems and *modus operandi* of North American general practice.

A fascinating problem in the free-enterprise countries is that of the evolution and growth of many sickness insurance schemes. In studying these I was particularly fortunate in being briefed by the president, Dr. George Baehr, and the staff of the Health Insurance Plan of New York: an organization which is a good example of many such schemes in the United States, now attempting to indemnify the patient against the present high costs of illness.

A PARADOXICAL SITUATION

Great Britain is a country where, as the result of a national 'insurance' scheme, a high proportion of the population is in effect fully 'covered' for treatment, whereas this is not so in the United States, although considerable attempts are being made by private enterprise schemes to produce such cover for as many of the population as wish to have it. Thus we in Britain can claim to be ahead, in this matter of comprehensive cover for the patient. Private enterprise schemes seldom achieve full cover for reasons to be discussed later. Whether North America is ahead of us technically is not within the scope of this article. In all fairness the answer probably is yes. From this it might be inferred that, under good conditions, treatment of the patient in the United States can be of a very high order. In spite of this, time and time again I found that many complained of, and were really concerned about, the high cost of medical treatment. Doctors, on the other hand, were not vocal about their complaints—if they had any. Thus, in comparing Great Britain and the United States, we have a paradoxical situation where the doctors in Great Britain tend to be dissatisfied, and the patients seem reasonably pleased with the treatment they are receiving, whereas, broadly speaking, in the United States the reverse is true.

THE COST

In the United States, a general practitioner's surgery visit costs from 3 to 5 dollars (21*s.* to 35*s.*), a home visit £2 10*s.* to £3 10*s.* The cost of living in the United States, however, is two to three times as high as our own, and the expense ratio of many doctors is inevitably higher than in this

country. Equipment is often considerably more ambitious than we ourselves would require or could afford to own. It is predominantly because of this high cost of medicine that the trend towards greater insurance is gathering impetus and public opinion is forcing the doctors to accept the participation of more and more patients in such schemes. It must be realized that the North American is naturally and historically far more independent and resistant to government pressure and organization than we are in Great Britain. He is far less inclined to trust rule by government or to accept any encroachment upon his legitimate preserves. Thus, whilst it is unlikely that a national health service, as we know it, is imminent in North America, very considerable efforts are being made to develop satisfactory medical insurance arrangements, and there is now a number of varying schemes for patients to choose from.

About 90 million of the 150 million inhabitants of the United States now carry some form of insurance to help to pay hospital charges. The best known of these is the Blue Cross scheme. It usually, but not always, pays hospital costs in full in a private or semi-private two- to four-bedded room. It also pays for x-ray facilities, pathological tests, theatre charges, drugs and anæsthetics. In some instances, particularly maternity care, it may pay only fixed charges, the patient being required to make up the difference. Other insurances provide part-payment schemes, and schemes which do not pay for the first two or three days in hospital, thus discouraging too ready entry to hospital. About 74 million people carry some form of insurance against doctors' bills: as a rule, only for surgical or medical care in hospital.

This means that around 50 per cent. of all expenses for hospital care throughout the United States are now covered by insurance schemes, but protection against doctors' charges is much less adequate: perhaps of the order of 15 to 20 per cent. Only a small fraction are insured against day-to-day medical expenses outside hospital, although in the United States 90 per cent. of all medical care takes place outside hospital.

Insurance schemes themselves tend to produce inflationary charges. Companies do not allow sufficient amounts for operations or treatment expenses, and the surgeons add something to the figure allowed, thus laying themselves open to criticism on the grounds that they are adding something to a figure already considered to be adequate. This is a frequent occurrence, and tends to defeat the whole object of the insurance. Further, anæsthetists' fees and other charges may not be covered by such insurance schemes. When one considers that operation fees vary from several hundred dollars up to, perhaps, 5000 dollars in exceptional situations, it can be understood that in the United States the fear of the cost of illness is not by any means eliminated by insurance.

THE NEW YORK SCHEME

A brief description of the Health Insurance Plan of greater New York (HIP), as an example of one of the best schemes in operation, will show that ener-

getic work and research have in fact been carried out in the United States for a number of years to devise methods of providing comprehensive cover together with a high standard of treatment. It was interesting, if a little ironical, to find that many of the methods adopted and snags encountered have been basically similar to our own.

The HIP Scheme was started around 1947 as the culmination of twenty-

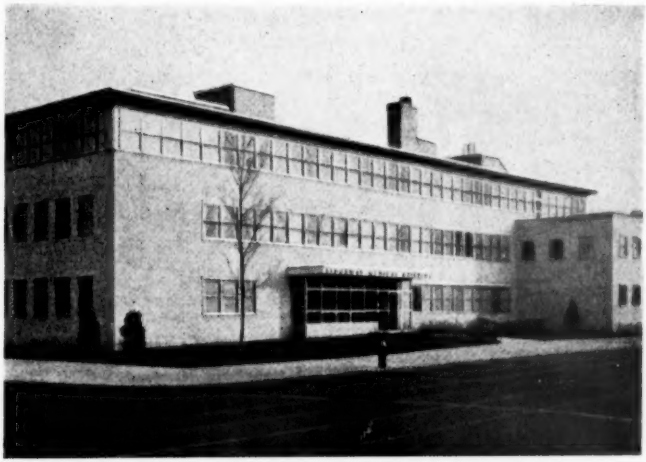

FIG. 1.—A Health Insurance Plan Medical Centre.

five years of experiment in medical care by Dr. George Baehr, its president. He pioneered in the field of treatment for people of moderate and minimal subsistence incomes, attempting to organize family doctor care for these people on a contract basis. The ground first chosen, in 1938, was the families in a housing development scheme in Eastern New York called 'Vladeck' houses. The average income of these families was $1,200, which entitled them in any case to free care in public hospitals and clinics as medical indigents. For one dollar a month per household, family doctor care, besides the hospital care already provided, was organized.

Immediately, however, a great outcry arose from neighbouring doctors that their private practice would be invaded, but in due course peace was restored after much negotiation and discussion. With the cooperation of Mr. La Guardia, the famous Mayor of New York, and later, loans from the Rockefeller foundation and others (since repaid), over thirty medical groups, located in various parts of the city, have been set up. Those enrolled as patients to date, are all members of employment groups and unions such as the chefs and cooks union, city employees, other firms and organizations.

The essence of the scheme is its endeavour to encourage the formation of medical treatment groups within the city with their own premises (fig. 1, 2, 3) fully equipped for a wide range of treatment of a general and specialist

character. Stress is laid on the fact that all those attending must be first seen by, and must remain under the supervision of, a physician or general practitioner, even though they may be receiving specialist treatment. Encouragement is given to the concept of early treatment and detection of disease, as in our own system.

HIP is a community-sponsored, non-profit organization operating under

FIG. 2.—A waiting room in a HIP medical centre.

the supervision of the State Department of Insurance. All those enrolled must also be insured by Blue Cross or other hospital insurance scheme. The annual premium is about $45 for a single person, and of the order of $130 for a family of three or more. Those earning above a certain figure, i.e. $5000 for an individual, pay 20 per cent. more. The average premium received by HIP is about $36. In many cases the cost is shared by the employer. Patients are entitled to unlimited care, and the average number of items of service rendered by the doctor per patient is between five and six per annum. Each medical group receives a capitation fee per annum for each patient of $29.4. As this figure includes the cost of any ancillary services received by the patient, it cannot be compared with our own capitation figure in any way. Pathological, x-ray, physiotherapy, and home nursing facilities are also covered.

So much for the outline and ideals of the Scheme. Difficulties have been encountered in its organization, and resourceful methods for dealing with some of these have been adopted. Open antagonism from reactionary colleagues was experienced by those who pioneered groups. Premiums for such a scheme were relatively high, and employers had to be encouraged to contribute towards part payment of premiums. Abuse of the service by patients has not been a difficulty or a problem, as the items of service figures

will show. There has been some competition from commercial insurance organizations putting forward attractive-looking schemes for covering major medical catastrophies only, in which the patient may reasonably, if mistakenly, acquiesce.

SUPERVISION AND REMUNERATION OF DOCTORS

It is interesting to see an attempt being made to ensure that the quality of medical care is kept up to a high standard in the groups by a system of

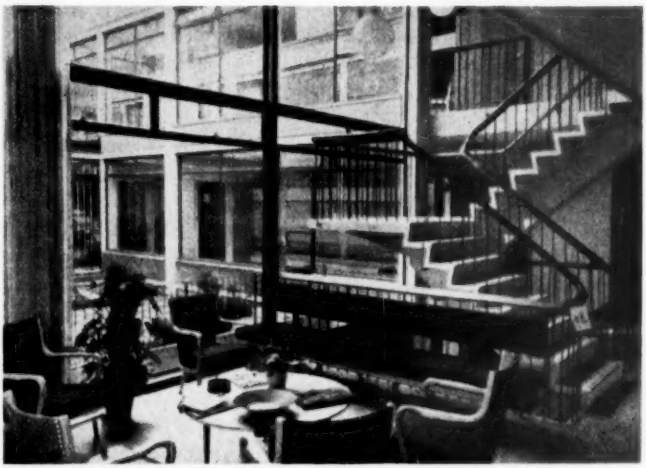

FIG. 3.—Another example of a waiting room in a HIP medical centre.

supervision. This is carried out periodically by a team of senior men of recognized eminence and repute. Such a system calls for the utmost care and delicacy in handling, but it was claimed that, as a result, the work of certain groups or individuals had been improved over a period of time. This method of checking the quality of medical care, although experimental, seemed to be searching and comprehensive, covering matters such as clinical investigations carried out, conclusions reached and final results. Such an assay of treatment, and the work of the doctors, appeared to be far more exacting and searching than anything we in Great Britain are used to. It is met by criticism from certain doctors but I was told that many of the practitioners involved regarded it as a challenge to inefficiency.

Each group is paid capitation fees according to the number of patients it handles. This amount is divided up to cover expenses and the payment of the individual doctors. Attempts are made to maintain a parity of income status between the various types of doctors within the groups, whether specializing or not, as it is considered that in group practices healthier relationships are likely to be preserved if those participating receive com-

parable payment. With such an aim, the estimation of the relative importance of the role of each doctor will not suffer.

PAYING FOR WHAT YOU GET

It is felt from discussion with many in the United States, that the average American likes to pay for what he gets, and is extremely interested in whether he is receiving good value. If he is satisfied about this, he is relatively conscientious and ready to pay. Many middle-class people and young couples live up to the hilt of their incomes, having multiple payments such as hire purchase to cope with. Thus, weekly insurance premiums fall into line with their pattern of living. In the United States there is a current saying that you have to be very rich or very poor if you wish to receive satisfactory treatment.

The standard of equipment and facilities for treatment varies but in medicine, as in most things, the American of today is fastidious. He tends to be inquisitive and keen to receive a high standard of treatment in so far as it is within his power to do so. We must not forget that the equipment and amenities of some of the clinics mentioned are of a first-class standard, and that the majority of American practitioners have regular hospital appointments and contacts, with the encouragement towards the development of a special field that this gives. Despite the contract method of treatment in such schemes, both the patient and the doctor are fully aware that everything the patient receives, he is paying for without subsidy.

It must also be remembered that the American is possessed of a considerable sentimental and charitable streak, and may well be encouraged to take a practical interest in the misfortunes of his sick fellow-creatures undergoing hospital or other treatment by voluntary contributions and even in assisting with the nursing of patients in hospital.

SUMMING UP

It is felt that the development of schemes such as HIP is of interest to ourselves and should be studied. Other outstanding examples of group practice prepayment plans are the Kaiser Permanente Foundation plan in San Francisco and the Ross Loos Clinic in Los Angeles. Altogether in the United States, over 5 million people are receiving prepayment medical care of a comprehensive scope from such organizations comparable with HIP. Whilst any attempt to produce in effect a National Health Service in the United States will continue to be strenuously resisted by the doctors, considerable progress is being made in providing self-supporting diagnostic and treatment centres of a high order of efficiency. To a great extent the advent of a National Health Service is being prevented by the formation of organizations of insurance and groups as described, run on a self-supporting basis and possessing a wide range of diagnostic and treatment facilities.

I am indebted to Dr. George Baehr and the staff of the Health Insurance Plan of greater New York, for all the help they gave me.

GENERAL PRACTITIONERS' FORUM

ANÆSTHESIA IN DOMICILIARY OBSTETRICS

By M. W. FORDHAM, M.R.C.S., L.R.C.P., D.Obst.R.C.O.G.
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The question of anæsthesia in domiciliary practice is a vexed one: every anæsthetic agent and method of administration has its advocate. Whatever the argument for or against, the fact remains that without the correct equipment what should be an interesting and successful manœuvre may prove a source of embarrassment and frustration.

There is no agent without its difficulties or dangers, especially in view of the fact that one is so often dealing with an unprepared patient, probably with a stomach full of glucose water or sweet tea. The three most commonly used agents are chloroform, trichlorethylene ('trilene') and ether. This article is intended to outline some of the difficulties that are encountered and to explain in more detail the method which I now use and some of the equipment that it entails. Before proceeding further, I would state emphatically that, because of the danger to the fœtus and the possibility of maternal laryngeal spasm, intravenous thiopentone has no place in domiciliary midwifery.

CHOICE OF ANÆSTHETIC

Chloroform.—This I have often seen administered most successfully with or without ether as a mixture. From the point of view of the obstetrician it is a good anæsthetic on account of its rapidity of action and the excellent relaxation usually obtained. It must, however, be given by someone experienced in its technique and, on the whole, I find that there are very few of the younger doctors who have had the experience. There is, of course, the inevitable bogey of liver damage. For these reasons I believe that it will be used less and less. For my part I never employ it as I do not consider myself sufficiently well versed in its use. I would also add that I have delivered more shocked and flaccid babies after chloroform has been administered than with any other anæsthetic agent.

Trichlorethylene.—Given in the 'cyprane' inhaler, trichlorethylene is excellent as an analgesic in the first and second stages of labour: and has the advantage of being more portable than gas and air apparatus. Some doctors, using this inhaler at high concentrations, perform low forceps at the outlet most successfully: for my part I have been severely discouraged from this technique by vivid memories of finishing up upon two occasions with a restless and excitable patient.

Ether.—This, I am sure, is the anæsthetic of choice as it is (a) safe, even in unskilled hands, especially in the unprepared patient, (b) there are few doctors who have not had considerable experience with it. Its disadvantages

are: (a) it is unpleasant to inhale, (b) the length of time usually required for the induction, (c) its inflammability. Ether can always be administered by the 'rag and bottle' method although this often takes a long time. Nevertheless, I am confident that this is the anæsthetic of choice in semi-skilled hands given to the patient who has not been prepared and has a stomach full of liquid.

APPARATUS

In the past I was so dissatisfied with the usual methods available in general practice for administering anæsthetics, and being also aware of the desirability

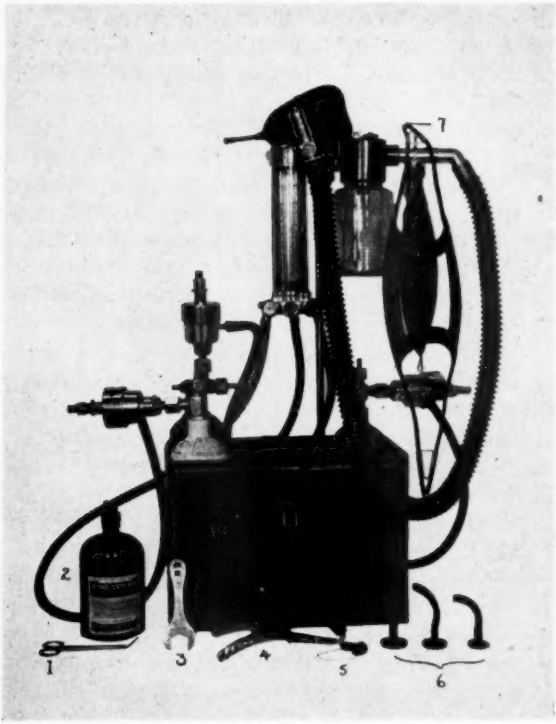

FIG. 1.—The Boyle's apparatus assembled for use: (1) Moynihan tongue forceps. (2) Bottle of ether. (3) Spanner for Adams valves. (4) Fergusson gag. (5) Spare washers for Adams valves, threaded on string. (6) Guedel airways (three sizes). (7) Connell harness.

of having oxygen readily available, that I decided to experiment with other methods. I was most fortunate in being able to purchase an old portable Boyle's apparatus, and with the help of Messrs. Coxeter & Son Ltd., I was

able to convert this into a more modern piece of equipment (fig. 1). The size of the case (fig. 2) for carrying it in is 14½ by 17½ by 8 inches (37 × 44 × 20 cm.), and the total weight, including full cylinders of oxygen, nitrous oxide and carbon dioxide, is 44 lb. (20 kg.). There is thus no difficulty in finding room for it in the smallest of cars.

From the patient's point of view it is far pleasanter to receive the ether by this method rather than the 'rag and bottle' and from the doctor's angle it is a tremendous comfort to realize that oxygen and a rebreathing bag are readily to hand. On two occasions when I have encountered fœtal distress in the fully dilated patient, and was unable to obtain the immediate assistance of another doctor, it was possible to induce the patient, strap on the mask with a Connell harness after inserting a Guedel airway and proceed with the forceps delivery leaving the midwife in charge of the patient, happy in the knowledge that oxygen was 'on tap'. On each occasion the other doctor, who had been called, arrived before the delivery was completed and was able to take charge of the already anæsthetized patient; meanwhile valuable time had not been lost.

FIG. 2.—The Boyle's apparatus packed up in the case for transporting.

It should be added that trichlorethylene can also be used with this apparatus.

In addition, this apparatus has many other uses: on several occasions it has been used as a source of oxygen to resuscitate a new-born baby. It can be used for minor general work in the surgery or in the home—e.g. for whitlows and incising breast abscesses—and cases of cardiac asthma and coronary occlusion have been glad of the immediate source of oxygen until some more permanent arrangement could be made. I know that it may be argued that it is an expensive piece of equipment, but I am sure that any busy practice would soon find that it pays for itself, and more, within a short time, apart from its very real use and, above all, the confidence that the practitioner would have in its safety and simplicity.

FURTHER EQUIPMENT

In these days of advances in hæmatology, no practitioner should undertake the care of the expectant mother without tests for blood grouping, Wasser-

mann and Kahn reactions and hæmoglobin estimation. Apart from assessing any rhesus incompatibility, knowledge of the patient's blood group saves a great deal of time in cases in which the Flying Squad has been called out.

Among my equipment (fig. 3) I always carry an infusion-giving set, a bottle of 'plasmosan' and a wooden board measuring 39 by $5\frac{1}{2}$ by $\frac{1}{3}$ inches (99 × 14 × 1.5 cm.) for splinting the arm. In the event of a post-partum

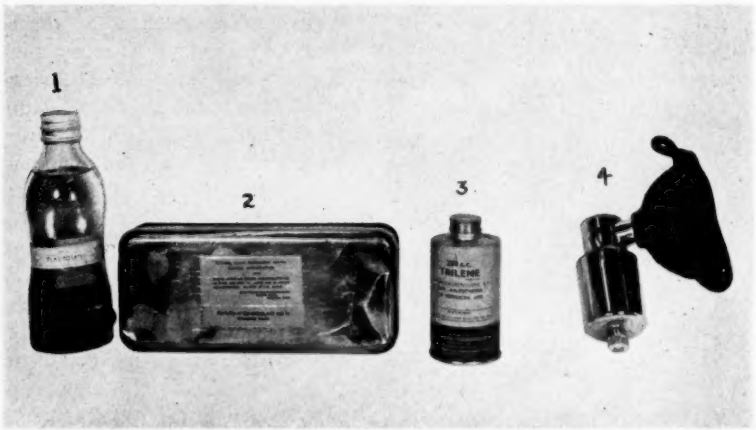

FIG. 3.—Additional equipment: (1) Bottle of 'plasmosan'. (2) Infusion-giving set. (3) Small can of trichlorethylene. (4) 'Cyprane' inhaler with mask and key.

hæmorrhage, while some worthy relative is telephoning for the Flying Squad, the midwife and myself put up a 'plasmosan' infusion, having first taken a sample of blood into a tube. When the Flying Squad arrives, the serum already taken from the patient is cross-matched against the stored blood, which then replaces the 'plasmosan'. In this way time is saved and, while awaiting the arrival of the blood, the intravenous fluids being administered combat further shock to a large extent.

SUMMARY

Today the tendency is for more and more mothers to have their babies in institutions. If the general practitioner hopes to continue to be responsible for confinements in the home, he must be prepared to keep abreast of modern advances and to have by his side equipment which will give the maximum comfort to the patient and which is capable of directly assisting him in dealing with any reasonable emergency.

This article has advocated the use of ether as the most useful anæsthetic agent, and that in the interests of ease and safety it should be administered through a portable Boyle's apparatus. Suggestions have also been put forward of other purposes for which this apparatus can prove most useful and life-saving.

A brief outline of infusion materials has also been added. These can be readily carried and come within the orbit of the anæsthetist-practitioner.

I am indebted to Mr. R. G. Mason, F.I.B.P., of the photographic unit, Stoke Mandeville Hospital, for the photographs.

FINANCE AND THE YOUNG GENERAL PRACTITIONER

By D. HUTCHISON, M.B., Ch.B.

Musselburgh, Midlothian

On entering general practice the young doctor will need to have an idea of financial budgeting. On returning from National Service he should have some savings, which may increase as an assistant, but will more than likely disappear 'into the red' when he becomes a junior partner. This state of affairs may be modified if a budget is drawn up and plans are made in advance. As no-one can be the 'average' doctor, I give my own figures and findings modified by the experiences of friends similarly situated in large semi-rural or semi-industrial practices. At times 'round figures' have been used for simplicity but I do not think these will give a false impression. It is assumed that there is only limited capital available.

THE FIELD

There is no doubt that too many doctors are seeking to enter the present vacancies in general practice, and the conditions and salary offered may therefore be very poor. Contracts are difficult to obtain and 'gentlemen's agreements' are all too common and unsatisfactory. The usual method of entering general practice is as an assistant, or perhaps a 'trainee' assistant, later becoming a junior partner. The average gross financial reward of the posts advertised in the medical press is £1,100 to £1,300 as assistants. The figures given in the following budget are taken from life, and a young general practitioner will find that they must not be exceeded if he is to remain solvent.

On nationalization the 'goodwill' of a practice was bought out but this payment has yet to be made and will not be made until death or retirement. In the meantime the practitioner's money is bringing in about half the interest it could give and the value of every pound has been halved. No payment is made for the use of doctors' surgeries but if they use a health centre may pay £1 a day. There are no tax concessions for any improvements made in surgery premises which must have passed an approved

inspection. The more spent for the good of his patients the less there is for the practitioner. Principals are not able to do everything they might wish for their assistants as there is a narrow financial range beyond which they cannot afford to go. On the whole, £1 a year for each patient may be usual, and, if the number of patients is known, a quick estimate of the gross income may be made. Of this, 35 per cent. is estimated as expenses. It must be remembered that partnership shares are usually on the gross takings and if the average expenses are, say, £700, not much is left out of £1,200 but much more out of £3,600. If one decides to 'squat' in an area an inducement grant may be payable if there are too few doctors to the population, this decision being taken by the Executive Council, but a better idea of conditions may be obtained from the Local Medical Committee.

FINANCIAL SECURITY

A certain amount of *capital* must be gathered and the more the better. This may be obtained from relatives without interest, or from other sources at a high rate of interest, or it may be earned before entering the field of general practice. All must join the Forces and if a short service commission is taken on call-up it is possible to serve one extra year and receive £150 for each year, free of income tax. While in the Forces one may undertake surgeries for local doctors and receive fees, or do a locum in leave periods. Alternatively, there are the more highly paid posts abroad, often with little opportunity to do anything but save!

Compulsory *superannuation* contributions are deducted at source: 6 per cent. of the net salary (about £60). The eventual benefits are related to the sum total of the net National Health Service payments and it therefore follows that in the earlier years of service the resulting pension and so on are hardly worth taking into account. If one has dependents or debts, other provision must be made for accident, sickness, or death.

A *life assurance* of £1000 may cost £25 a year, but it is better to get a combined accident, sickness and life policy, the cost of which will vary with the benefits offered. For some £50 a year it is possible to obtain a life assurance of £1000, and accident and sickness cover giving £12 12*s*. a week for 26 weeks, and then half that sum to the age of 65 in the event of prolonged disability. When it is considered that twice this amount would be required to cover the cost of a locum and any outstanding debts, it will be realized that £100 spent on this form of insurance would not be too much, but the young doctor will not be able to afford this. It is well to take expert advice on insurance problems, remembering that insurance is a good form of 'compulsory' saving.

In addition, £17 is payable each year as an assistant, and £22 as a partner, for *National Insurance* stamps. If desired, superannuation contributions may be paid while in the Forces, at the rate of the last N.H.S. post held before call-up. This may require about £100 from capital.

THE CAR

A car is not absolutely essential, but it allows a far wider selection of possible appointments. It is, in fact, often the first question asked at an interview, being given precedence over one's medical qualifications for the post. The car would need to be reliable, give many miles per gallon, and be of reasonable appearance and price. Rather than risk a cheaper, second-hand model of unknown ancestry, I bought a new 'Morris 1000' at £625.

Under present hire-purchase regulations, the minimum deposit is one-third of the cost, with two years for repayment of the balance. I did not have unlimited capital so I decided to pay £300 cash and the balance in twenty-four monthly payments of £18. This meant that I had now allotted £216 of my salary for a year for the car for two years. In addition there would be running expenses of a further £200 a year: insurance (£20), road tax (£12 10*s*.), petrol and oil, servicing and repairs. Finally, there is depreciation, which has worked out at £100 a year, as after two years' hard use over some 35,000 miles the repair bills begin to increase alarmingly. It has therefore paid me to trade in my old car in part exchange for a new one. It will be noticed that our total is now around £500 and yet most advertised posts offer £100 or £150 a year for car expenses.

SUBSCRIPTIONS

One must be a member of a medical defence society in this litiginous age (£2) and may join the British Medical Association (£6 6*s*.) as well as other professional societies. Then there are subscriptions for periodicals and the replacement of books and so on, for which it is as well to set aside about £25.

HOUSE PURCHASE AND MAINTENANCE

After a year as an assistant in any practice, the hope of a partnership should be fulfilled, or it is time to move elsewhere. If the intention is to remain, however, the biggest step yet faced has now to be taken—house purchase, as one can rarely get a house to rent. The size of any loans and the resulting interest to be paid must be limited. I was advised that the maximum loan should never be more than three times my gross annual salary and that my bank overdraft, included in this loan, should not be more than a year's gross salary. Interest repayments will work out at between £6 and £7 a year for each £100 borrowed. Eighty per cent. of the purchase price may be obtained by loan from a building society, and repayments may be made over a period of 20 years.

A doctor's house must suit the practice, rather than the doctor. It must be big enough to set aside rooms for consultations, and this usually means too big. It must be in the most advantageous situation and district and, finally, it must be the right price. A house costing £3000 will require £600 from capital and the repayment of the loan will be about £175 a year for 20 years, more or less, depending upon variations in the interest rate. From capital

will also have to come a further sum for decorating and furnishing, say £300. The annual house budget will include loan repayment, £175; property tax, £25; rates, £60; repairs and renewals, £30 (these may be small in the first years or even put off for a little but this inevitably means a much bigger outlay later on and is false economy); heating and lighting, £50; total £340. It may also be necessary to allow about £25 for telephone bills. These are actual figures but do not take any account of tax relief.

On joining a practice one has to take over a share of the property and fittings, as well as outstanding fees. This may well involve a capital outlay of £500.

DOMESTIC BUDGET

Next there is the cost of every-day living, with its wide variations: housekeeping, domestic assistance, furniture renewals, clothes, holidays and recreation, and the like, total £500 or more a year.

BANK OVERDRAFT

By this stage one may well be indebted to the bank, interest being between £7 and £8 on each £100. An assistant is paid monthly, but a partner every three months. It is vital therefore to have all financial commitments planned out and falling due at different periods. In January, road, property and income taxes are due as well as rates, so it is advisable not to have insurance premiums due at this time.

INCOME TAX

An expert accountant must, of course, be employed, but in addition, it is as well to take legal advice on one's contract of service, as upon this depend many tax reliefs. Minor alteration in the wording may gain major tax concessions for the assistant while costing the practice no more money.

It should be remembered that *expenses* for income tax relief must be reasonable, and actually incurred exclusively for the performance of the duties of the employment. Capital expenses incurred to put one in a position to begin to carry out these duties are not allowable. As an assistant, income tax is deducted at source under Schedule E, but as a partner it is payable half-yearly under Schedule D. Under the latter, tax reliefs are based upon the previous year's figures. It therefore follows that in the first year as a partner there are no previous year's expenses to give any tax relief, and tax will be high.

Car tax allowances include 40 per cent. of the initial cost and a 25 per cent. annual allowance on the cost in the first year, and on the written-down value thereafter. Relief on other expenses is less the proportion incurred in private use. It is difficult to convince the tax commissioners that an assistant who has a contract for '£150 or £100 car expenses' actually spends more

than this. It is better for the contract to give the total remuneration 'to include car expenses'.

Superannuation and part of *life assurance* payments are allowed for tax relief but not those for accident and sickness, or national insurance. Renewals and replacements of *library and personal equipment* are allowed but not their original capital outlay.

The doctor's *house* is normally used for practice purposes and this proportion is allowed: often half the rent and rates, as the rooms used are usually on the ground floor in the best situation. Expenditure on structural alterations to make surgery accommodation is not allowable for tax relief. The ratio of other shared house-practice expenses will have to be agreed upon, and the surgery share allowed: heating, lighting, domestic service, cleaning, decorating, furniture, front garden assistance, and so on.

A *doctor's wife* will have to do much work for the practice and she may be paid up to £3 10*s*. a week free of tax. Over this amount there will be tax deductions. The payment must in fact be made and be reasonable for the work performed. Club and charity *subscriptions* are not allowable even if they are of great indirect benefit to the practice. *Entertainment* expenses may be allowed if hospitality is combined with professional advice. Payment by an assistant for *board and lodgings* is not allowable for tax relief, but if they are provided by the practice and a correspondingly reduced salary is received, then there is benefit to the assistant without extra cost to the practice.

THE BALANCE

	Annual Budget	Capital
The car	£500	£300
Security	£130	
Subscriptions	£30	
Domestic	£500	
Income tax	£100	
As an Assistant:	£1,260	with £300 from capital
House	£385	£900
As a Partner:	£1,645	with £1,700 from capital

CONCLUSION

In conclusion, there are two factors which may change the present pattern completely. First, the result of the present remuneration claim, and secondly, in 1958, doctors may retire on pension having served ten years in the National Health Service. If there is a substantial rise in remuneration more doctors will be in a financial position to afford to retire, and there will then be more opportunities for young general practitioners.

SECTION OF GENERAL PRACTICE OF THE

ROYAL SOCIETY OF MEDICINE

DISCUSSION ON THE BREAST IN PREGNANCY AND LACTATION

December 18, 1957

Dr. Mavis Gunther (*University College Hospital*).—The management of breast feeding is a technical subject which needs to be learnt by doctors and midwives, for with present-day knowledge most of the troubles of lactation can be prevented. It also needs to be learnt and understood because it is a fact that most mothers in England now do not know how to feed their first babies. In them the instinctive know-how which must have existed in previous ages is gone.

THE IMPORTANCE OF INSTINCT

Since instinctive actions in animals, such as nest building, can be lost to the adult bird if reared without a nest, the failure in humans may perhaps be attributable to present-day standards of modesty which deny the chance of mimicry. The baby's part in breast feeding fits more clearly into recent concepts about instinct. The delightful studies which have been made by Lorenz, Tinbergen and other workers have revealed a number of characteristics of instinctive behaviour which hold good throughout much of the animal kingdom. Instinctive behaviour has been found to be evoked usually by a specific pattern of stimuli, usually working within one sensory system; a shape seen, for instance, which calls up one instinctive response through an innate release mechanism. The pattern of stimuli, or sign stimulus, which drives a baby to feed is thought to be the physical shape of the tissues of the breast which fill the baby's mouth. The eagerness with which a full-term baby will feed can be predicted by measuring the protractile quality of the mother's areola and nipple. This instinctive response, like others, needs to be evoked fully at the right time of life—within the first three to four days—or the response may be much weaker. Once it has been called in action fully the baby's eagerness and skill in feeding are usually lastingly reinforced.

Experiment has shown that for feeding to be successful a well-formed nipple is useful but is far less important than the total length composed of nipple, areola and subjacent tissues which will enter the mouth past the jaw gap. At term these tissues should be capable of being drawn into the mouth for about 3 cm. after passing a 2-cm. gap and if in pregnancy there is any doubt about this protractility, Woolwich shields should be fitted. Dr.

Harold Waller estimated that 40 per cent. of English primiparæ needed this help.

The protractility of the nipple is more effective if the baby is correctly positioned, with the lower jaw driving hard at the breast. The antenatal care and the after-delivery correct positioning bring the added reward that they save the mother from developing the commonest kind of sore nipple.

The engorgement of the breasts in the early puerperium can be largely avoided by giving mothers minute doses of stilbœstrol, 2 tablets, each of 0.25 mg., being taken an hour or two after delivery and one tablet a day later.

ACUTE INTRAMAMMARY MASTITIS

Acute intramammary mastitis appears to be a two-stage disease, the first consisting of milk retention in an area of the breast; this can be completely resolved at this stage by emptying the retained secretion. In the second stage, after about two days, emptying is commonly no longer possible and infection almost inevitable. The misleading feature of the first stage is the fever which it often causes, and the suggestion has been made that the rise in temperature, which may be accompanied by a rigor, is caused by absorption of pyrogenic substances from the retained milk or as a neurogenic effect from a distended tissue rather than from bacteriæmia.

Because of the time limit on the possibility of emptying, and therefore of complete resolution, treatment is urgent. It consists of an antibiotic cover as, if infection comes, it will always be staphylococcal in origin, and of emptying of the breasts by the baby, by hand or most easily by the 'humalcator'. Feeding should be continued usually for two days; if the condition is not then improving lactation must be suppressed. If infection comes lactation should be suppressed so far as possible before incising, otherwise fresh areas of unemptied breast tissue extend the area of mastitis and in turn await infection.

Prevention requires that breasts should be kept empty whenever the secretion is liable to be greater than the baby's demands. Above all, breasts must be kept empty while lactation is being suppressed.

Dr. David Morris (*British Hospital for Mothers and Babies, Woolwich*).—The preparatory techniques for breast feeding, introduced by Dr. Waller at The British Hospital for Mothers and Babies at Woolwich, have taught some of the factors which impede or aid successful lactation.

THE IMPORTANCE OF ANTENATAL CARE

Routine antenatal examination of the breasts ensures the early diagnosis of such anatomical abnormalities as retracted nipples and their treatment with glass or plastic shells. Likewise, the detection of inelasticity of the skin forewarns the possibility of engorgement and its prevention by the early use of stilbœstrol. Manual expression, taught antenatally and practised during the last four to six weeks of pregnancy, enables mothers to express

surplus milk without difficulty after the baby has fed and thereby avoids rising tension and inhibition of further milk flow. In such conditions as prematurity or birth injury, lactation can be established and maintained in spite of the baby's condition.

Breast feeding in present-day society appears to be a task of some difficulty, requiring help, guidance and encouraging support during the lying-in period. Many of the failures of breast feeding occur due to a lack of adequate supervision and preparatory training. The cracked nipple detected before skin erosion occurs is a common example of an avoidable cause of failure of breast feeding.

The recent survey carried out at Woolwich (Morris and Jeffereys) in conjunction with the sociology department of Bedford College illustrates what can be achieved. In contrast to the national trend of 43 per cent. of women fully breast feeding at eight weeks, 69 per cent. of women discharged from hospital fully breast feeding were still doing so at three months, and 46 per cent. at six months.

CAUSES OF FAILURE

Little is still known of the cause of failure of lactation in those apparently successfully breast feeding but it may well be that social trends and the ready recourse to bottle feeding are largely responsible. The increasing tendency to introduce early mixed feeding may also be an important factor. Dr. Welbourne's study, 'Bottle Feeding: A Problem of Modern Civilization', among the Baganda tribe in Uganda illustrates the influence of the availability of bottles and dried milk. In a period of five years she found an increase from 14 per cent. to 30 per cent. of supplementary bottle feeds before six months, with a significantly higher incidence of respiratory and bowel disturbances in those bottle fed. The weight gains were 1 to 2 lb. (0.5 to 1 kg.) higher in those fully breast fed.

It is too quickly assumed that women do not wish to breast feed and, unless pregnant mothers are helped antenatally and during the lying-in period, they may well abandon breast feeding without giving themselves a fair trial. The old reasons for advocating breast feeding—avoidance of respiratory and bowel infections—are no longer tenable. But recent work (Sinclair, 1957) and the increasing knowledge of the psychological importance to both mother and child necessitate a closer critical view than is at present held.

References

Morris, D., and Jeffereys, M.: 'Breast Feeding, Living Conditions and Health' (in the press).

Sinclair, H. M. (1957): *Brit. med. J.*, **ii**, 1424.

Welbourne, H. F.: 'Bottle Feeding: A Problem of Modern Civilization' (in the press).

Dr. Joan Ferreira (*Middlesex County Council*).—My point of view has developed while working in maternity and child welfare clinics. For fifty

years these clinics have been trying to promote breast feeding, but in spite of this we find it steadily declining. For example, Ross and Herdon (1951), at Bristol, found that the incidence of babies breast fed for three months fell from 77 per cent. to 36 per cent. between 1929 and 1949. During this period the proportion of babies attending welfare centres rose from 27 per cent. to 83 per cent. The number of babies leaving hospital fully breast fed has also dropped, falling from 86 per cent. in 1950 to 76 per cent. in 1955. Reports of various surveys show at least one-third of mothers breast feeding at three months. In groups having special help, two-thirds or more were doing so. It looks as though there is a middle third which is liable to be influenced either way by the quality of the preparation and care given.

How might welfare clinics have done better? The teaching given to mothers could have been more imaginative and effective. Perhaps the advice given has been at times behind current medical opinion (Wickes, 1952). There may have been pressure to continue unsatisfactory breast feeding when efforts to improve the situation had not succeeded. An anxious mother and frustrated baby will harm the attitude of other people towards lactation.

MOTHERING THE MOTHER

A woman's emotions are often unstable during the first month after childbirth, and in order to mother her baby properly she needs mothering herself. Many babies seem to be taken off the breast unnecessarily during this period because of undue anxiety about their progress, misplaced fears about the condition of the mother and want of a little encouragement to persevere. Health visitors and midwives could do much to prevent this, but only if they are working with the support of the family doctor. There should be no break in the care of the hospital-confined mothers after they get home, and the health visitor should make herself readily available. If possible, she should get to know the mothers in the antenatal period. Her visits should be at times when they can be most effective and the whole family must be taken into account, for they are all influencing the outcome of lactation. It is important that the health visitor or midwife should appreciate the high quality of the care needed, particularly in the case of first babies, when the pattern is being set for the rest of the family.

REPLANNING ANTENATAL CLINICS

The numbers attending local authority antenatal clinics have dropped sharply since the introduction of the National Health Service, so that clinical care of the breast in pregnancy is a responsibility which is passing elsewhere. It has been suggested that the time is coming when patients will cease to attend for clinical examination and that the clinics will turn into centres where expectant mothers can learn about the coming confinement and care of the baby. There is a special need for mothercraft teaching today because, with smaller families and women working outside the home, girls grow up having little idea of what will be required of them.

At present, mothercraft classes are held in many clinics by midwives and health visitors but on the whole only a few interested women attend. More work should go into the planning of this teaching, which has to be personal, understanding and, to some extent, experimental. It should be supported by those members of the medical profession best able to help. Common difficulties and early discomforts in breast feeding could be discussed, and attitudes towards it ventilated. The pleasure rather than the duty of suckling should be brought out.

Many factors will have influenced the performance of the breast in lactation before pregnancy starts. The woman's heredity (Wickes and Curwen, 1957), her feeding experience in infancy, her growth, nutrition and education and the example of her own mother will have shaped her abilities and outlook.

SOCIAL CLASS DIFFERENCES

Several workers have shown a social class difference in breast-feeding performance. This may emphasize the need for freedom from domestic worries (Westropp, 1953), and the importance of good diet, ability to cope and the belief that breast feeding is best (Hytten and Thomson, 1955). I have met an attitude, occurring more particularly in women of social class IV and V, that breast feeding is 'not quite nice'. Is this a lingering-on of the Victorian ideal of refined womanhood which has seeped slowly through society, or just a way of expressing failure to breast-feed? Many of the women appear to be complacent about not fulfilling their feminine functions and seem to give up breast feeding without regret. There is incomplete understanding of women's sexual emotions, and more knowledge of them might lead to greater insight into problems of lactation. Newton (1955) has pointed out the relationship between a woman's attitude to menstruation, sexual intercourse, labour, breast feeding, and infant care.

Some women feel that suckling their babies is one of the things they expect from life. Mothers who have fed their babies successfully always want to do it again. I am sure that helping a woman to nurse her baby is important to the well-being of both.

There are many ways in which more could be done to encourage lactation. I have only touched on some of the ways which come within the orbit of the local health services.

References

Hytten, F. E., and Thomson, A. M. (1955): *Brit. med. J.*, **ii**, 232.
Newton, N. (1955): 'Maternal Emotions', New York.
Ross, I. A., and Herdon, G. (1951): *Lancet*, **i**, 630.
Westropp, C. (1953): *Brit. med. J.*, **i**, 138.
Wickes, I. G. (1952): *Ibid.*, **ii**, 1178.
——, and Curwen, M. P. (1957): *Ibid.*, **ii**, 381.

CURRENT THERAPEUTICS

CXXII—BARRIER CREAMS

By I. B. SNEDDON, M.B., F.R.C.P.

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To most people barrier creams mean substances applied to the skin before work which will prevent damage to the skin by irritants handled at work. In recent years the scope of barrier creams has increased to include prevention of bedsores and of ulceration of the skin by discharges from intestinal fistulæ and colostomies. Barrier creams also have a place in the treatment of skin conditions due to abnormal sensitivity to sunlight and they have penetrated into the home where they are advocated as a means of avoiding the rough, red and ugly housewife's hands.

INTRODUCTION

Any measure which can reduce the incidence of dermatitis in industry is desired not only by the medical profession but also by the employers of labour to whom the wastage of trained personnel from dermatitis may be a severe financial loss. The idea that the application of a barrier cream to the skin before exposing it to industrial hazards would reduce the incidence of dermatitis was therefore welcomed and widely accepted. Not only is the procedure supported by legislation, for in certain industries the employer is compelled by law to provide 'a suitable barrier cream or similar protective substance', but official advice on their use has been given by the factory department of the Ministry of Labour and National Service in several memoranda. Added to this, common law proceedings for negligence may succeed if barrier creams have not been provided by an employer. This combination of fear and the intelligent salesmanship of the manufacturers of barrier creams is more responsible for their widespread use than their proven efficacy. Their true worth is still a matter for hot debate and their main virtue is that they make the task of cleaning the skin at the end of the day's work easier, and therefore there is less trauma from abrasive cleaners and detergents.

There are many explanations of why they fail. Barrier creams have been used for purposes for which they were not intended. Even if the correct barrier cream has been applied, too much has been expected of it and its protective action may have lasted only an hour or so and the workers have not been instructed to re-apply it several times a shift. Then, there are certain occupations which necessitate constant immersion of the hands in alkaline solutions or industrial solvents for which at present there is no adequate protection. The present position was summed up admirably by

the editors of the 'Year Book of Dermatology, 1955-56', when they said: 'It is evident that no effective all-purpose protective ointment has been developed. While those available have some practical application, their usefulness unfortunately is limited'.

A belief in the effectiveness of barrier creams may lead to neglect of other more important steps which may be taken to prevent dermatitis. A high standard of cleanliness of the workshop, adequate protective clothing and prevention of contact with potential irritants and sensitizers by other means than barrier creams are more likely to lower the dermatitis rate.

Barrier creams may themselves cause dermatitis; particularly is this so when they are used beneath gloves. Goldsmith and Hellier (1954) reported that, in testing one protective cream, a skin reaction occurred constantly when it was applied under an occlusive dressing although it did not do so when left uncovered. Tzanck *et al.* (1952) believe that at times barrier creams may facilitate the penetration of noxious substances into the deeper layers of the skin. They observed positive patch-test reactions to mixtures of barrier creams and various potential irritants although each substance tested separately gave a negative reaction. Horner (1948) issued a warning against the dangers of incorporating animal fats in barrier creams used against substances such as T.N.T., nitrobenzene and aniline compounds, as their absorption may be increased rather than prevented. She also pointed out (Horner, 1950) that when used against carcinogens such as pitch, coal tar and mineral oil, the barrier should not increase the risk of penetration. It can be seen therefore that in order to be effective and indeed harmless the appropriate barrier cream must be carefully selected for the particular purpose for which it was intended.

TYPES OF BARRIER CREAM

The basic requirements of barrier creams, according to Schwartz *et al.* (1957) are:—

(1) They should be non-irritating and non-sensitizing.
(2) They should offer actual protection from the irritant as shown by laboratory tests.
(3) They should be such good cosmetic preparations that they can be easily applied and feel pleasant to the skin.
(4) They should be easily removable by ordinary soap and water and yet be so adherent that they will not easily rub off under actual working conditions.

To this list, Horner (1950) adds the important property that they should have a pH near to that of normal skin, 5.5 to 6.5.

Barrier creams in current use are of three main types:—

A dust barrier which is used to protect the skin against sensitizing dusts and against substances which if absorbed will produce systemic poisoning.

A water-repellent barrier for those whose hands are constantly exposed to water, alkalis, and water-soluble oils.

An oil-repellent or water-miscible barrier which is a protectant against oils, greases and solvents and which facilitates their removal without the use of abrasives or oil solvents.

The barrier substances subcommittee of the B.P. Codex Revision Committee published, in 1955, the following formulæ:—

Dust barrier:	
Casein (Rennet, finely powdered)	3
Sodium alginate	2
Glycerin	6
Stearic acid	10
Triethanolamine	1.55
Chlorocresol	0.2
Phenol	0.5
Distilled water	to 100
Water-miscible barrier:	
Kaolin (sterilized)	20
Fuller's earth (200 mesh)	3
Hard soap (powdered)	12
Glycerin	6
Stearic acid	2
Sodium chloride	1
Chlorocresol	0.2
Phenol	0.5
Distilled water	to 100
Water-repellent barrier:	
Hard paraffin	25
Soft paraffin	11.75
Liquid paraffin	3.5
Cetostearyl alcohol	5
Triethanolamine	0.7
Stearic acid	1.8
Chlorocresol	0.2
Distilled water	to 100
Skin-conditioning cream:	
Monostearin, self-emulsifying	10
Glycerin	6
Triethanolamine ricinoleate	1
Wool alcohols	6
Chlorocresol	0.2
Oleyl alcohol	3
Distilled water	to 100

These are now being widely used. The skin-conditioning cream is used after work if the hands are dry and degreased.

One disadvantage of these formulæ, which has been reported to me, is that the water-repellent and water-miscible barriers both have an unpleasant smell which tends to persist even after several washings of the hands. Although a perfume could be incorporated, it is difficult to find one which is a non-sensitizer and which does not increase the price excessively.

In the last few years *silicones*, which are water repellent, have been increasingly used in barrier creams. Silicones are organic compounds of silicon with a structure of alternate atoms of silicone and oxygen with organic groups, such as methyl and phenyl, attached to the silicone atoms. These compounds produce a durable, flexible and stable water-repellent film on the surface of articles and on the skin. Talbot *et al.* (1951) reported on the value of 30 per cent. silicone oils in a petrolatum base in the treatment of a variety of dermatoses, mainly around sinuses and colostomies

and claimed successful control or cure of 58 out of 61 cases. Since that time a number of silicone barrier creams has become available, but their initial promise has not been fulfilled in practice.

EVALUATION OF BARRIER CREAMS

There have been many attempts to test the efficiency of barrier creams in the laboratory. Sadler and Marriott (1946) devised an ingenious method by spreading the barrier cream on a glass surface and allowing it to dry. The film was then immersed in the liquid against which it was supposed to be a barrier and the time estimated for it to disintegrate was measured. They also described a technique for estimating the permeability of a film of barrier cream. They found the tests useful since those creams which failed on the tests also failed when used in the factory. Cumming *et al.* (1947) also carried out tests with protective creams against T.N.T. and tetryl, estimating the degree of penetration of a film of barrier rubbed into the skin. As a result of these tests they were able to develop a more effective cream.

Cruickshank (1948) used the fluorescence of oil in ultra-violet light as an indication of degree of penetration of the barrier cream by machine oil. He investigated six soapy base creams, nine vanishing creams and six greasy base creams. His conclusions were that certain soapy base creams might be of some value. Vanishing creams were unlikely to be of any value and the greasy base creams might assist in the removal of oil from the skin. In a further experiment to indicate how long protection was likely to be effective, the soapy base creams designed to resist oil penetration were found to permit complete penetration in one hour.

Smith *et al.* (1953), working with silicone (dimethylpolysiloxane) nitrocellulose and castor oil in a greaseless vanishing-cream base, estimated the protective effect against allergens in a group of subjects known to be sensitive to the allergen. Thirty of the 36 subjects demonstrated varying degrees of protection. It was found that, generally speaking, the greater number of days the silicone cream was applied prior to testing, the greater its protective efficiency. The cream was also compared with three other preparations, one of which contained 30 per cent. silicone in petrolatum. The silicone creams were superior to the non-silicone creams. That protection against heavy oils is still impracticable was shown by Shaw and Crowe (1955), who compared new silicone preparations with a number of other barrier creams against the action of soap and water, soluble cutting oil and heavy cutting oils. The preparation containing 30 per cent. silicone in petrolatum was the best protective, being twice as effective as petrolatum alone, but none of the preparations could withstand two minutes' exposure to three heavy cutting oils or a petroleum solvent.

In an attempt to test silicone protective cream, Morris (1954) applied patch tests on both arms of subjects known to be sensitive to the patch-test substances. One arm had previously been treated for seven to eleven days

with silicone creams. In 20 tests, six were more positive on the silicone protected arm, seven were more positive on the unprotected arm, five showed no difference and two were negative on both. Three of the patients returned to work after using the silicone barrier cream for ten days and suffered an immediate recurrence of their eruption.

It can thus be seen that, even in laboratory tests, barrier creams have given very conflicting results and that, so far as oils are concerned, no satisfactory protective has been found. It is surprising that, despite universal use of barrier cream, there have been such scanty reports of field trials in a factory. There has been a number of claims by enthusiastic industrial medical officers, notably Bourne (1944, 1954), but the fact of having an efficient industrial medical officer may lessen the incidence of dermatitis and there is no account of a true controlled trial. There are considerable difficulties in devising such a trial since, without strict discipline, it is not possible to avoid the errors which would result from workers not applying barrier creams regularly and thoroughly, but there is a great need for such a trial in this country. A recent report from the United States described the use of a cream of 52.5 per cent silicone fluid in a bentonite base in an aircraft engine factory (Suskind, 1954). This contains a far higher percentage of silicone than the creams available at present and would be expensive.

The cream was available to 146 workers and 114 used it regularly or periodically; 55 had a history of skin trouble while at their job and 44 had active skin disease of mild type when the experiments started. The substances which the workers handled were numerous and included rust preventive in oil, turbine oils, degreasing solvents, aircraft fuels, xylene, machine-tool coolants, cutting oils, synthetic detergents, sodium silicate and paraffin. During five months of observation no case of sensitivity to the cream was seen. Some complained of dryness of the skin and this, together with slight vesicle formation in those who wore rubber gloves, was ascribed to sweat retention caused by the silicone film. Of the 44 who had mild dermatitis, 24 recovered while using the cream. No benefit from this cream was seen in those who had their hands in paraffin and water-glass or who were exposed to trauma of the skin.

It was also noticed that those who had particularly dirty and greasy jobs were able to clean their hands more easily. The creams were removed by an anionic synthetic detergent, sodium lauryl sulphoacetate.

BARRIER CREAMS IN MEDICINE

The silicone barrier creams have been recommended for the prevention of napkin rashes and particularly for the treatment of the skin around colostomy and ileostomy openings. They should be used immediately after the operation and before the skin has become damaged.

Bateman (1956) reported the beneficial effect of a 20 per cent. polydimethyl siloxane cream used for routine treatment of the backs of geriatric patients in the prevention of bedsores. The method cuts down nursing time and is said to give better results than the traditional use of soap and water followed by spirit.

More recently, Roberts and Williams (1957) have commented favourably on the use of a 20 per cent. silicone cream as a prophylactic against bedsores in bedridden elderly patients treated by district nurses. They claimed

that the technique is quicker and easier and that the results are better than with traditional methods. Both Bateman and Roberts and Williams advocate the giving of vitamin C, 50 mg., and a vasodilator, tolazoline, 25 mg. thrice daily, in addition to the silicone cream so that from their results it is not possible to be sure that the silicone cream alone would be equally effective.

LIGHT BARRIER CREAMS

Protection of the skin against sunlight may be a necessity in those who suffer from the various forms of actinic light dermatitis, chronic discoid lupus erythematosus and xeroderma pigmentosum. The discovery of the light-protecting powers of mepacrine and its related compounds has not done away entirely with the need for a protective cream which can be applied to the exposed skin surfaces.

The properties of such a cream have been laid down by Smithers and Wood (1952):

(1) Good screening power.
(2) Easy to apply.
(3) Good covering power.
(4) Moderately water resistant.
(5) Allow, but not be removed by, perspiration.
(6) Not easily rubbed off but not too difficult to remove.
(7) A finish which could be cosmetically acceptable.

Countless substances have a sunscreening effect and preliminary tests of their efficiency can be carried out spectrophotometrically. The ultimate test can only be made on the skin. A simple, though not cosmetically desirable, sunscreen is yellow petroleum jelly. Commonly used substances are derivatives of *para*-aminobenzoic acid, phenyl or menthyl salicylates. One of these may be combined with titanium dioxide, a colouring agent, and a vanishing-cream base. A modification of the following formula used by Smithers and Wood is available commercially:—

Cream	
Titanium dioxide	25.0 per cent.
Magnesium stearate	12.5 ,,
Butyl stearate	12.5 ,,
Menthyl salicylate	20.0 ,,
Pigment base	2.0 ,,
Vanishing cream	28.0 ,,
Pigment base	
Burnt umber	50.0 ,,
Red ferric oxide	25.0 ,,
Yellow ferric oxide	25.0 ,,
Vanishing cream	
Stearic acid	15.0 ,,
Cetyl alcohol	1.0 ,,
Triethanolamine	1.5 ,,
Diethyleneglycol monoethylether	10.0 ,,
Distilled water	72.5 ,,

METHOD OF APPLICATION OF BARRIER CREAMS

The hands should be clean and dry before applying a barrier cream. The cream should be applied sparingly to the whole of the hands, and not just rubbed into the palms as is so often done. It is much more important to coat the backs of the hands, paying particular attention to the sides of the fingers and the webs between. The cream should be well rubbed into the nail folds and under the free edges of the nails. If used against solvents a coating of cream should be applied several times during a shift.

Ideally, silicone cream should be applied twice daily for ten days before exposing the skin to irritants; when the protective film has been formed, a once daily application will be sufficient to maintain protection.

SUMMARY

Barrier creams are of value in certain circumstances but, as yet, there is no universal protective.

In many industries the skin trauma is so great that no cream can be expected to provide protection.

Oil is not a potent sensitizer, but much damage is done to the skin by the methods commonly used by workers to cleanse themselves.

Barrier creams will aid cleansing and in this way they are of some value.

The silicone creams have been shown to be generally superior to other forms of barrier creams but they still do not answer the problem of prevention of dermatitis, and they are expensive. They have a place in the prevention of skin maceration from intestinal discharges and in the incontinent patient.

References

Bateman, F. J. A. (1956): *Brit. med. J.*, **i**, 554.
Bourne, L. B. (1944): *Ibid.*, **ii**, 290.
—— (1954): *Ibid.*, **ii**, 1954.
Cruickshank, C. N. D. (1948): *Brit. J. industr. Med.*, **5**, 204.
Cumming, W. M., Cameron, M. C., Cumming, E. B., and Macraild, M. C. (1947): *Ibid.*, **4**, 237.
Goldsmith, W. N., and Hellier, F. F. (1954): 'Recent Advances in Dermatology', 2nd ed. London, p. 327.
Horner, S. (1948): *Pharm. J.*, **160**, 7.
—— (1950): *The Practitioner*, **164**, 234.
Morris, G. E., (1954): *Arch. industr. Hyg.*, **9**, 194.
Roberts, G. W., and Williams, F. M. (1957): *Nursing Times*, **53**, 1206.
Sadler, C. G. A., and Marriott, R. H. (1946): *Brit. med. J.*, **ii**, 769.
Schwartz, L., Tulipan, L., and Birmingham, D. J. (1957): 'Occupational Diseases of the Skin', London, p. 110.
Shaw, J. M., and Crowe, F. W. (1955): *Arch. Derm. Syph. (Chicago)*, **71**, 379.
Smith, C., Day, T. L., and Zimmermann, E. H. (1953): *J. invest. Derm.*, **21**, 111.
Smithers, D. W., and Wood, J. H. (1952): *Lancet*, **i**, 945.
Suskind, R. R. (1954): *Arch. industr. Hyg.*, **9**, 101.
Talbot, J. R., MacGregor, J. K., and Crowe, F. W. (1951): *J. invest. Derm.*, **17**, 125.
Tzanck, A., Sidi, E., Albahary, C., and Longueville, R. (1952): *Arch. Mal. prof.*, **13**, 153.
'Year Book of Dermatology, 1955-56' (1956): Editorial comment, Chicago, p. 112.

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MINOR SURGICAL PROCEDURES

II.—SUPERFICIAL WOUNDS

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The term 'superficial wound' covers a variety of types of wound involving the skin and subcutaneous tissues. The 'wound' may be an abrasion, an incised wound, a laceration or a puncture wound. Although these types of injury have some features in common, each may involve the risk of particular complications and each may require different treatment.

COMPLICATIONS

Superficial wounds are extremely common, especially in children; many are treated in the home and never seen by a doctor. Even the most apparently trivial, however, are liable to complications, and if these occur the doctor may be consulted at a late stage. When a doctor sees a superficial wound at an early stage an important and inescapable part of his responsibility is to foresee, and if possible to prevent, any complications.

INVOLVEMENT OF DEEPER STRUCTURES

Although the involvement of any important structure under the subcutaneous tissue means that the wound is not really superficial, the possibility that the wound is only apparently superficial must first be considered. The real practical importance of this can best be emphasized by examples. A tiny puncture wound in a finger, as by a pin or a needle, may have penetrated and infected a tendon sheath. A small incised wound at the wrist or elbow, as by a fragment of glass, may have injured or even divided a nerve. A tiny flying fragment of metal penetrating the skin near a major vessel may leave a trivial mark in the skin but, if it hits the artery, may be the cause of a subsequent traumatic aneurysm or arterio-venous aneurysm. A small wound in a critical situation may involve a joint cavity and the danger of infective arthritis.

These possibilities must always be considered—more or less seriously according to the situation of the wound and the importance of immediately subjacent anatomical structures. Inevitably, on seeing the wound, the doctor will start from such anatomical premises. In all cases, however, he should obtain, from the patient or from any reliable witness, as detailed a statement as possible of the cause of the injury. What caused the wound? Was it clean or dirty? What degree of force was applied? Was there much bleeding? Careful examination will exclude nerve injury but it may be impossible to be sure whether a tendon sheath, a joint or a major artery has sustained minimal damage. If there is doubt the doctor will have to use

his judgment whether to adopt full prophylactic measures or simply to arrange to see the patient again soon and regularly until he can be sure that no complications are threatening.

HÆMORRHAGE

Bleeding from a superficial wound may be profuse, especially if the injury is to a vascular part like the face or scalp. A cut on the side of a finger may divide the digital artery. Such bleeding is more alarming than dangerous and is readily controlled by local pressure. Wounds in some situations, such as the face or the finger pulp, may be difficult to bandage firmly enough to arrest bleeding from a small open artery but digital pressure kept applied over the dressing for a few minutes will control the bleeding until the vessel end is sealed by clot. Any small artery in the wound may start to bleed again when the wound is cleansed for suture and may then require control, either by a fine ligature or by a suture underrunning the vessel.

PYOGENIC INFECTION

The most likely complication is infection by pyogenic organisms. The risk of this must be estimated first by obtaining as much information as possible about the condition of the object that caused the wound and about the cleanliness of the patient's skin. The character of the wound will be noted: the danger of infection is greater in the case of a lacerated wound with devitalized tissue or in a penetrating injury in which contaminating organisms have been carried under intact skin. If the injury is seen at a later stage the signs of established inflammation may be present.

TETANUS

Although this is a very infrequent complication of superficial wounds it is a notoriously serious one. There is more danger of infection by the tetanus bacillus if the wound is contaminated by soil or road dirt. The infection has more chance of becoming established if the wound is a penetrating or lacerated one, because the bacillus grows in anaerobic conditions. The size of the wound is of much less importance: there would, for example, be much more risk of tetanus in a small puncture wound of the foot caused by standing on a dirty nail than in an extensive clean incised wound on the forearm.

It is probably neither practicable nor desirable to give prophylactic treatment for tetanus in every case of superficial wound. The administration of anti-tetanic serum is not without risk; sensitivity reactions are frequent and cases of fatal anaphylactic shock have occurred. Prophylaxis by giving a 'booster dose' of tetanus toxoid is much safer and is fortunately becoming more common, but this method is applicable at the time when a wound is sustained, only if the patient has previously been immunized with toxoid. In that case a 'booster dose' of toxoid must be given instead of anti-tetanic serum.

If a doctor is asked to treat a superficial wound one of his first responsibilities is to decide whether appropriate prophylactic treatment against tetanus is to be given. If the character of the wound or of the contamination suggests that there may be some danger of tetanus he must not hesitate to do so. But if the wound is judged to be quite superficial, without devitalized tissue in its depth and probably uncontaminated as well as easily cleaned, he may rightly decide not to give such prophylactic treatment. In this case, to protect himself against the totally unexpected, he should make a written note on the record that he has deliberately omitted this treatment: e.g. 'It was considered advisable *not* to give A.T.S.'.

If prophylaxis is deliberately omitted in the initial stage, it should none the less be given later if, after a day or two, the wound shows signs of deep inflammation.

If prophylaxis is to be given, and if the patient has not previously had toxoid, an injection of 3000 international units of anti-tetanic serum is injected intramuscularly. Before doing this, it is advisable to test the patient for sensitivity by giving an intradermal injection of 0.2 ml. of the undiluted serum. If an urticarial reaction or a zone of erythema develops round the injection in the next thirty minutes the patient should be considered sensitive and should be gradually desensitized by giving graded doses of the antitoxin. This procedure is unavoidably time-consuming and a general practitioner who has observed evidence of sensitivity in a patient will usually have to refer the patient to the casualty department of a hospital. The graded doses are given intramuscularly at fifteen-minute intervals as follows (the standard serum contains 3000 I.U. in 1 ml.):—

Dilution of A.T.S.	Volume of injection
1 in 10	0.5 ml.
1 in 10	1.0 ml.
1 in 5	0.5 ml.
1 in 5	1.0 ml.
Undiluted	0.1 ml.
,,	0.2 ml.
,,	0.4 ml.
,,	0.4 ml.

If any untoward reaction is observed the progress must be more gradual. Occasionally, even with these careful measures a delayed mild anaphylactic reaction is seen after an hour or two. Unfortunately, a severe anaphylactic reaction sometimes occurs in response to the full dose of serum even after an intradermal test dose has given a negative reaction. In view of these dangers the doctor who gives anti-tetanic serum should have beside him a syringe charged with 0.5 ml. of adrenaline solution, 1/1000, for intravenous injection if signs of sweating and collapse present. This may be followed by parenteral administration of an antihistamine drug.

When the patient has had previous immunization with tetanus toxoid the correct prophylactic treatment is to give a 'booster dose' (1 ml.) of the toxoid intramuscularly. No precautions are necessary, but it must be

emphasized that this measure is useless unless the patient has had previous immunization.

RETAINED FOREIGN BODY AND INGRAINING OF DIRT

Even in apparently trivial wounds there may be danger of a foreign body being driven into the subcutaneous tissues. If the injury has been caused by a broken stick, small pieces of the wood may have broken off; if an object has penetrated the clothes before injuring the skin, it may have pushed a small fragment of material under the skin. If the foreign body is overlooked infection is certain to occur and the danger of tetanus is materially increased. When a retained foreign body is suspected the depths of the wound should be carefully explored with appropriate instruments.

When the injury is an abrasion caused by a dirty object or surface, the danger of ingraining of dirt must be considered. This is particularly important on skin surfaces normally exposed and in women who will be concerned about the cosmetic results. The skin may heal over the dirt with the result that there is an indelible tattoo mark such as is seen typically in the healed superficial injuries of coalminers. This 'tattooing' can be prevented only by vigorous scrubbing with a firm brush and in some cases, as when a woman's face has been deeply abraded on a gritty road, there should be no hesitation in advising general anæsthesia for this treatment.

SCARRING

In some situations, and particularly on the face and on the pulp of the finger, the danger of a disfiguring or a tender scar should be considered. If there is no loss of tissue, scarring should be minimized by meticulous toilet and suture of the wound. In some cases there is loss of tissue. Any sizable fragment of skin which has been cut off the nose or the finger pulp should be washed and preserved, if possible, in a solution of penicillin in a sterile test-tube until it can be sutured on again. This may be achieved with success even after a delay of some hours. If there is loss of tissue from such a critical site, and if the fragment is lost, the patient should be sent to a surgeon who will probably replace the lost tissue with a free graft of skin from behind the ear.

FIRST-AID TREATMENT

If a sterile dressing such as surgical gauze is not immediately available, the wound can be covered with clean cotton-wool or a well-laundered piece of cloth (linen or cotton). The first object will be the arrest of bleeding, which can always be effected by gentle pressure. The dressing may be held in position until a suitable bandage is available.

If appropriate surgical treatment is not immediately available, steps should be taken to effect a preliminary cleansing of the skin in the vicinity of the wound and the wound itself. It is best first to clean the skin round the wound while the dressing is held in position with the fingers. This cleansing may be done with soap and water or with a soapy disinfectant such

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as cetrimide or other quaternary ammonium compound (e.g. 'cetavlon', 'roccal'), or a chloroxylenol in soapy solution (e.g. 'dettol'). If bleeding has been arrested the dressing can then be removed and the wound itself gently cleaned with a similar mild antiseptic; a fresh supply of the antiseptic should be used for application to the wound. The wound should then be re-covered with a dry dressing of sterilized gauze or 'tulle gras', or, if these are not available, by a piece of clean cloth. A firm but not tight bandage is applied. Measures such as these will usually be employed before the patient is seen by a doctor.

FIG. 1.—Method of closing a small superficial wound with adhesive strapping.

CHOICE OF DEFINITIVE TREATMENT

The doctor who subsequently treats the wound will remove the first-aid dressing to inspect the lesion. He may decide that it is a trivial abrasion or cut which does not require further treatment except re-cleansing with antiseptic and reapplication of a sterile dry dressing or 'tulle gras'. More serious lesions will require treatment according to the type of injury. Some superficial injuries are so severe that they must be treated in hospital: e.g. extensive incised wounds or lacerations which involve much devitalization of skin.

ASEPTIC TECHNIQUE

If further treatment is required mere 'no touch' technique will not usually suffice as an aseptic precaution. Full operating-theatre routine will not be required except for lesions which by reason of their extent or complications require treatment in hospital, but the doctor who undertakes to suture a superficial wound in his own surgery or in the outpatient casualty department of a hospital must adopt meticulous, if less elaborate, precautions against infection. All dressings, lotions, sutures and instruments must be sterile. Outdoor clothing should be covered by a clean laundered coat or gown and a mask should be worn. The hands and forearms should be thoroughly washed with soap and water but without undue scrubbing by a brush, except for the nails and the harder skin of the hand. The hands and forearms should then be completely dried with a sterile towel. This drying is essential to guard against drips of water running from the fingers down instruments into the wound. Drying with a clean laundered towel is preferable to no drying at all. It may be completed by application of surgical spirits to the hands. The wound area must be properly cleansed with antiseptic and 'towelled off' so that there is no contamination from surrounding regions. It is particularly important to see that the length of suture

material used does not trail in a non-sterile area while the suture is being inserted and tied.

ABRASIONS

Superficial abrasions, however trivial, should be carefully cleansed to remove all dirt. This is best done with a mild antiseptic solution such as cetrimide which will not cause undue pain. The importance of removing ingrained dirt, especially on the face, has already been mentioned. The surface should then be dried with sterile gauze. An extensive abrasion may be dressed with 'tulle gras', covered by dry gauze and cotton-wool and bandaged firmly. Only a single layer of 'tulle gras' should be used, because the object is to permit drainage through the interstices of these dressings and absorption of discharges by the overlying dry dressings. These dressings are left undisturbed for five to seven days, unless pain or systemic upset suggests that the lesion is becoming inflamed.

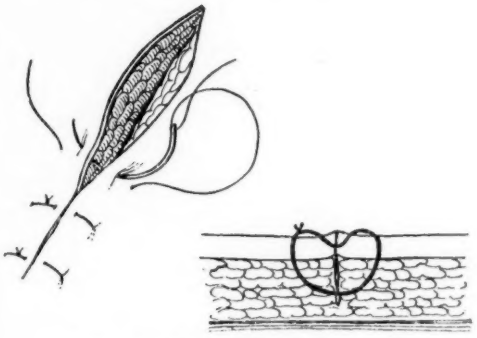

FIG. 2.—'Vertical mattress' suture.

Abrasions on exposed surfaces such as the face, or on the knees in children, are better treated by exposure. They are allowed to dry and to scab after adequate cleansing.

The local application of antibiotics or sulphonamides to skin lesions is not advised but, if evidence of invasive infection develops, appropriate oral or parenteral chemotherapy should be given.

INCISED WOUNDS

Small cuts which are gaping slightly may be closed without suture by using a piece of adhesive strapping. This strapping should be shaped with a narrow neck which crosses the wound; the wound itself is in this way left partly exposed so that it is kept dry by exposure (fig. 1).

A cut wound should be sutured only if it is reasonably clean. If there is evidence that it is likely to be heavily infected it is better left open and closed later by delayed primary suture. This can be conveniently achieved by inserting appropriate sutures immediately and tying them to close the wound two or three days later.

If suture is contemplated an injection of local anæsthetic will usually be required. A solution of 1 per cent. procaine or 1 per cent. lignocaine is used. In the case of a clean wound this may be injected under the skin by introducing the needle through the cut surfaces. A fine (hypodermic) needle

should be used and small quantities of the solution placed appropriately should be sufficient.

Sutures may be of any appropriate material, but linen or silk thread treated by some waxing process is most convenient. Sutures should be interrupted and placed as widely as is consistent with accurate apposition of the skin edges. Any tendency of the skin edges to invert can be obviated by using occasional vertical mattress sutures (fig. 2).

Special care must be used in suturing wounds which may lead to unsightly scars in exposed areas such as the face. It should be remembered that a needle hole caused by a clumsy cutting needle will leave a scar of its own which is cosmetically very undesirable. This may be avoided by using the finest cutting needles but, if these are not available, it is advisable to use a non-cutting needle on the face. An ordinary round-bodied sewing needle serves the purpose. A wound on the forehead may involve the underlying aponeurosis; the galea should be sutured with catgut to avoid a depressed scar.

LACERATED WOUNDS

A lacerated wound may have irregular edges and some devitalized tissue. A neat closure with good cosmetic result may be more difficult to obtain. The more extensive or more lacerated wounds may require hospital treatment. If the practitioner undertakes the closure of a small lacerated wound he should excise any obviously devitalized tissue with sharp scissors or a scalpel, but skin should be sacrificed only if it is very ragged or deeply discoloured. A lacerated wound may be more liable to infection than a cut by a clean object. If infection is expected penicillin powder may be insufflated into the wound before closure. Only a fine dusting of powder is required. (Penicillin powder B.P. contains sulphanilamide powder as an excipient.)

An adequately sutured wound should be covered with a dry gauze dressing. On the face or scalp it is better left exposed, or it may be sprayed with 'nobecutane' and then left exposed. If subsequently there is any threat of inflammation in a sutured wound the patient should be given an appropriate antibiotic orally or by injection. If frank sepsis occurs sutures should be removed to permit adequate drainage.

Sutures are removed after about four days from the face, scalp or neck. In other situations they may be removed after seven or eight days.

PUNCTURE WOUNDS

If there is any suspicion that a foreign body is retained in a penetrating or puncture wound the wound should be explored with anæsthesia. If there is reason to believe that a puncture wound is grossly contaminated the skin opening should be made wider by incision to ensure adequate drainage, and if evidence of inflammation develops appropriate antibiotic treatment should be started. A puncture wound will seldom call for any suturing.

IMMOBILIZATION

When an extensive superficial wound on a limb, or more particularly a wound in the region of a joint, has been treated surgically it may be advisable to immobilize the part temporarily, both to give the tissues the best possible conditions for reaction against infection and to ensure undisturbed primary healing. This immobilization may be obtained by applying generous cotton-wool padding and bandaging which extend well beyond the region of the wound. More rarely, temporary immobilization with plaster of Paris splintage is advisable. Rest of the wound area is required, particularly if there is risk of infection or evidence of invasive inflammation. When the inflammation is severe the patient may have to be treated by bed rest and elevation of the part to help to reduce reactionary œdema.

REVISION CORNER

LIENTERIC DIARRHŒA

LIENTERIC diarrhœa (from λειοζ—smooth, εντερον—intestine, and having nothing to do with λιεν—spleen) is the old-fashioned term applied to a particular type of diarrhœa in which the bowel actions occur particularly soon after taking food. It may be freely translated as 'a slipperiness of the guts'.

'GASTROGENOUS DIARRHŒA'

It occurs in two groups of conditions: one rare, the other by no means uncommon. In the former it is associated with disease of the stomach and in these has been called 'gastrogenous diarrhœa'. Most usually the gastric condition is one of mucosal atrophic change with achlorhydria. It is known that if achlorhydria is present the stomach empties very rapidly, and this is presumably the mechanism of the diarrhœa. It should be noted, however, that, although achlorhydria is relatively common, diarrhœa as a consequence is far less so and these cases are, in fact, rare. When they do occur the diarrhœa is readily checked by the administration of dilute hydrochloric acid B.P. in doses of 30 minims (2 ml.), well diluted with lemonade, with meals. Diarrhœa of this type is also seen occasionally in cases of carcinoma of the stomach, particularly when there is extensive infiltration; here again rapid gastric emptying is probably the mechanism. It was a case of this kind that first introduced me to the term 'lienteric diarrhœa' on a teaching round of Dr. C. E. Lakin at the Middlesex Hospital in 1932.

'NERVOUS DIARRHŒA'

The second, and more frequently seen, group of cases occurs in tense, highly strung, individuals in whom it tends to be episodic. Here it is simply

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a variety of the well-known 'nervous diarrhœa'. Most often in these patients the diarrhœa is an early morning occurrence, but in some it is prolonged during the rest of the day in a lienteric fashion. Rapid gastric emptying again no doubt plays its part but, in addition, there is a general hypermotility of the whole intestinal tract and an exaggeration of the normal gastro-colic reflex. Colicky pain preceding the actions is not usual but may occur; the stools are generally pale and porridgy but may be frankly soupy and they often contain recognizable particles of undigested food. Even when the condition is frequent and of long standing, loss of weight and other consequences of chronic diarrhœa are strikingly absent. In many cases, but by no means always, the patients recognize the relation of their symptoms to nervous tension.

In treatment, kaolin sometimes helps, but the most useful drug is codeine given as tablet of codeine phosphate B.P. In a brisk attack as much as ½ grain (30 mg.) four-hourly may be required, but often ¼ to ½ grain (15 to 30 mg.) once or twice daily is sufficient, and it can safely be prescribed in this dosage for long-term use. In mild cases sedation with small doses of amylobarbitone or phenobarbitone, with tablet of codeine phosphate in reserve for exacerbations, is the usual treatment. In these patients anxiety about the possible social embarrassments of the diarrhœa often exaggerates and perpetuates the symptoms, and once confidence in the efficacy of treatment is achieved, the mere carrying about of a few codeine phosphate tablets in the waistcoat pocket is sufficient to avert many attacks.

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THE TREATMENT OF DOG BITES

DOG bites are much more common than is generally realized. In 1956 no less than 176 cases of dog bites were treated in the casualty department of a large provincial hospital, but of these only two cases required admission. Both these patients were admitted because the face had been injured. The vast majority of cases of this common injury are therefore suitable for outpatient treatment, and can be safely treated by the general practitioner.

Because of the risk of the subsequent development of rabies, dog bites have a sinister reputation. Owing to the strict enforcement of quarantine laws for animals, however, rabies has been completely stamped out in Great Britain. The sinister reputation of dog bites is therefore no longer warranted in this country.

TREATMENT BY THE GENERAL PRACTITIONER

The most common wounds produced by dog bites consist of nothing more than a series of superficial puncture wounds produced by the dog's incisor teeth. These wounds usually only just penetrate the full thickness of the skin and often do not even reach the subcutaneous tissues. These wounds

are unimportant except when they involve the face. It is a tradition that dog bites should be treated by cauterization with carbolic acid or silver nitrate. This is the correct treatment of wounds which may be contaminated with the virus of rabies, but should no longer be practised in Great Britain where the risk of rabies is now non-existent. Cauterization of wounds results in necrosis of tissue at the sides of the wound and this increases the scarring resulting from the wound. All bites of the superficial puncture type, not involving the face, can be safely treated in the surgery. The wounds should be cleaned by gentle swabbing with hydrogen peroxide or cetrimide, after which the surrounding skin should be dried with sterile swabs and the area widely covered with a sterile dry dressing. These wounds only rarely require suturing.

It is advisable to give all cases a prophylactic injection of anti-tetanic serum (1,500 international units), taking the usual precautions against anaphylactic reactions. This is not because tetanus bacilli are normal inhabitants of dogs' mouths, but because dogs so often smell and nose at fæcal-contaminated material containing tetanus organisms. It is therefore very likely that the organisms will be on the dog's nose and teeth.

TREATMENT IN HOSPITAL

A less common type of wound consists of a deeper laceration involving subcutaneous tissue and muscle. These bites are usually on the legs, and mainly affect the calf. They may even result in actual avulsion and loss of tissue. All wounds of this type require full excision under general anæsthesia. Patients with this type of bite therefore require hospital treatment. The wound should be treated in exactly the same way as any other wound: that is to say, all dead and damaged tissue should be excised, all foreign material such as clothing removed, and all pockets deep to fascial planes opened up. If this procedure is carried out within a few hours of the infliction of the bite, before the wound has become infected, primary suture of the wound can be carried out.

Dog bites involving the face merit special mention both because of the damage which may result to the lips, eyelids and even the eyes, and also because they are remarkably common. This is due to the widespread habit of nursing dogs on the lap. Facial bites wherever they occur also result in unsightly scars unless properly treated. In order to minimize the scarring which will result, all facial bites, unless extremely trivial, require careful and accurate suturing after cleansing. In the case of bites of the lips and eyelids, this can only be done adequately under general anæsthesia, and these cases therefore require hospital treatment, preferably under the care of a plastic surgeon. The opinion of an ophthalmic surgeon should be obtained in all cases in which the bite has involved the eye.

COMPLICATIONS

In Great Britain the most common complication of dog bites is the development of pyogenic infection in the wounds. This occurs much less commonly

than would be expected and, in my opinion, the prophylactic use of antibiotics is not to be recommended, provided the wounds are treated early and carefully. In the case of wounds which become infected after treatment or which are frankly infected when first seen, treatment should be on exactly the same lines as for any infected wound, with the aid of antibiotics and the incision and drainage of infected pockets in relation to the wounds. Once the infection has subsided it may be possible to aid healing and minimize scarring by carrying out delayed suture of the wounds.

Serious complications which should always be kept well in mind are the development of tetanus and gas gangrene. Tetanus can almost always be prevented by the prophylactic use of anti-tetanic serum as already described, but, if either condition should develop, it must be treated vigorously on the usual lines. This will, of course, necessitate the admission of the patient to hospital.

RABIES

No description of the treatment of dog bites would be complete without mention of rabies. Although the disease has been eliminated from Great Britain and therefore need not be considered in the treatment of dog bites here, in most other countries of the world the disease is endemic and should therefore always be considered. Furthermore, occasional cases are seen in this country in patients who have been bitten by rabid dogs abroad.

Rabies is due to a neurotropic virus. After inoculation the virus travels to the central nervous system along the peripheral nerves. The incubation period varies greatly, but averages thirty days for bites on the face, forty days for the arm and sixty days for leg bites. Once rabies has declared itself, death inevitably follows. The only way to be certain of preserving the patient's life is to start prophylactic treatment as soon as possible after the receipt of the dog bite. This should consist of the treatment of the wound and vaccine therapy. The wound should be encouraged to bleed freely, after which it should be excised and cauterized with pure carbolic acid or silver nitrate. There is no specific drug treatment—no drug has been shown to have any effect on the virus of rabies.

All persons who have received a dog bite in a country where rabies is endemic should start prophylactic anti-rabic treatment at once. The biting animal should not be killed, but should be confined under observation. If it is alive after ten days, the patient is free from risk, and prophylactic treatment can be stopped.

Prophylactic vaccine therapy varies with the exact vaccine used, but usually consists of a daily injection of vaccine for about fourteen days. The mortality rate for persons treated with prophylactic vaccine is very low, being less than 0.5 per cent.

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NOTES AND QUERIES

Oral Penicillin

QUERY.—Can you give me some definite information on the relative efficiency of the various forms of oral penicillin? The claims of the manufacturers are not helpful in that they weigh the evidence in favour of their own product by stressing one particular virtue. The three forms that I am thinking of at present are benzathine penicillin, ammonium penicillin, and penicillin V. Ammonium penicillin is apparently cheaper and, if equally effective, would, for that reason, be my choice.

REPLY.—Penicillin given by mouth in any of these three forms is absorbed to a degree which varies greatly from patient to patient. It is therefore necessary for the standard dose to be relatively large, and for greater security it is often advisable to give the first dose by injection. Ammonium penicillin is a simple salt of benzylpenicillin (soluble penicillin) and has the same action as the potassium salt which is the usual form manufactured in this country. Doses of 400,000 to 600,000 units (250 to 375 mg.) given four-hourly usually give blood levels adequate for the treatment of conditions due to sensitive organisms. Benzathine penicillin is a relatively insoluble derivative of penicillin. It is more stable in the presence of moisture and has a less bitter flavour and is therefore the form used in the flavoured mixtures which are especially suitable for administration to children. It is doubtful if comparable doses produce higher blood levels than soluble penicillin although this has been claimed. Phenoxymethylpenicillin (penicillin V) is also relatively stable and less bitter than soluble penicillin and is also suitable for administration in a flavoured mixture. When given orally in comparable doses it produces higher but not greatly higher blood levels than the other forms of oral penicillin. It should be given to adults in doses of at least 125 to 250 mg. (equivalent to 200,000 to 400,000 units) of soluble penicillin and there is a tendency to adopt the higher of these doses as the standard one. Benzylpenicillin and its salts are significantly cheaper than the other two preparations when given in the above recommended doses.

Unless palatability is of primary importance, as in the treatment of young children, and provided the tablets are kept and dispensed in containers which prevent the access of moisture there seems no good reason to replace soluble penicillin by either benzathine penicillin or phenoxymethylpenicillin.

PROFESSOR E. J. WAYNE, M.D., F.R.C.P.

Penicillin Sensitivity

QUERY.—Patients often assert that they are sensitive to penicillin and that they have been advised not to have it. Is there a patch or other form of test which gives an indication whether it is safe to use penicillin in these cases, and if so what is the best method of testing?

REPLY.—Sensitivity to penicillin is undoubtedly on the increase, but sensitivity to this cheap, safe and still remarkably effective antibiotic should not, *per se*, be a bar to its further administration in any penicillin-sensitive patient. This will depend upon the character of the drug reaction produced by penicillin, the biochemical make-up of the patient concerned, and the malady to be treated, as well as the availability of alternative remedies, particularly when penicillin may be life-saving. As a rule urticarial, morbilliform, rubelliform, erythema nodosum-like and even serum-sickness types of reaction should not prevent the practitioner from giving his patient a further course of penicillin or even continuing the course in which such reactions arise. Should his patient be an atopic subject or should he have had an anaphylactoid reaction, an exfoliative or exudative dermatitis, then penicillin should be withheld, and an alternative drug should be given. The practitioner should remember that penicillin may be essential for certain diseases, e.g. acute and subacute bacterial endocarditis, in which the causal microbe may be resistant to all drugs except penicillin.

Patch tests are of little value to the practitioner in detecting penicillin-sensitive cases—as, for that matter, are scratch tests—and both types of test may themselves sensitize the patient if he is not already sensitive to the antibiotic. They should be left entirely to the scientific investigator, save for the detection of sensitivity to penicillin in an atopic subject. When the practitioner has to give urgent treatment with an antibiotic to a patient with a strong family history of asthma, hay fever or eczema, he should inject a few units of penicillin intradermally and watch the patient carefully for signs of any anaphylactoid reaction, as he would were he giving an injection of antitoxic serum to the same individual. If there is any such reaction, however slight, in such a patient, one of the tetracycline group of antibiotics or appropriate sulphonamide drug should be given instead. The inexperienced house officer would be well advised to consult his chief before embarking on any routine treatment with penicillin in these patients.

E. J. MOYNAHAN, F.R.C.P.

UNIVERSITY OF MICHIGAN DIGITIZED

Poliomyelitis Inoculations

QUERY (from a reader in India):—In his article in 'Advances in the Treatment of Virus Diseases' (*The Practitioner*, 1956, **177**, 422) Professor C. H. Stuart-Harris, states that 'British children have so far received only two doses of inactive vaccine and a third dose is known to be desirable at about six months after the earlier inoculation'. Last April I received some of the vaccine, and gave the first two injections. A further supply of vaccine will shortly be available but I gather that the authorities may be reluctant to let me use it for the purpose of giving my patients a third injection. I should therefore be grateful if you could let me know what the general consensus of authoritative opinion is on this point.

REPLY.—It is established that two doses of poliomyelitis vaccine given at an interval of four weeks will provide protection against infection. This is evidenced by the result of field trials and a study of the antibody response in the serum. That a third, or 'booster', dose given after an interval will still further increase the level of serum antibodies is also well established, and the third dose may be given at from six to twelve months after the first two. It is generally accepted therefore that, whilst two doses give protection, if there is sufficient vaccine available, a third dose after six months will further reinforce the immunity.

H. M. WALKER, O.B.E., M.D.

Amenorrhœa after Childbirth

QUERY.—I have a patient aged 28 whose first and only child was born in hospital in July 1954. Labour was induced owing to marked hypertension. There was considerable loss of blood and she says that her hæmoglobin was 50%; at any rate it was 60% on her return home. Breast feeding was stopped in September as her blood pressure started to rise again but has been normal since.

She had a normal period in October followed by amenorrhœa. In March 1955 I started a three months' course of ethinylœstradiol, 0.1 mg. daily for sixteen days, followed by 'amenorone', 4 tablets daily for five days. Owing to a misunderstanding only one month's course was taken. Nevertheless she had a period in March and in April, followed by amenorrhœa. She had a three months' course from July to September with a period each month and another in October but no more. She had another three months' course from January to March 1956, each with a period but followed by amenorrhœa. She then consulted a gynæcologist in September 1956 and had a diagnostic curettage and cervical cautery in October. He reported that curettage revealed no recognizable endometrium. He advised thyroid extract, ½ grain (30 mg.) daily for two months, and stilbœstrol, 1 mg. daily for three weeks, and after an interval of a further week to repeat the course, giving three courses in all. The thyroid was not continued beyond that time as she was very irritable and could not relax. There had been no bleeding, and the gynæcologist advised continuing the stilbœstrol but adding 'amenorone' for the last five days. There was no bleeding. In March 1957 she took ethinylœstradiol and 'amenorone' each month for six months with a period each month finishing in August. There has been no period since.

The external genitalia are normal, as is the hair distribution. She enjoys married life. She does not particularly want another child. She is healthy and not at all neurotic, but has to work for financial reasons, and is often overtired. Is treatment necessary at all or is amenorrhœa associated with likelihood of disease? Is she infertile? I am now worried about the carcinogenic effect of œstrogens and would very much appreciate your advice.

REPLY.—The onset of amenorrhœa after childbirth at which there was stress from toxæmia and post-partum hæmorrhage suggests that there has been some degree of pituitary damage. This hypothesis would fit in with the 'periods' following the use of œstrogens, showing that the endometrium is responsive. Presumably the damage to the pituitary has not been gross enough to produce a fully developed Simmonds's syndrome. But amenorrhœa may be the first symptom and the condition may remain stationary or progress.

Having tried various courses of œstrogen treatment there would seem to be no reason to continue it, although the risks of carcinogenesis are probably slight. Before considering any further treatment full investigation by an endocrinologist would seem indicated and would be valuable in reaching both a diagnosis and prognosis.

Fertility would be affected in the presence of amenorrhœa although ovulation independent of menstruation can always occur and a pregnancy follow.

KENNETH BOWES, M.D., M.S., F.R.C.S.

Generalized Hyperidrosis

QUERY (from a reader in India).—I have a patient, a lawyer, aged 42 years, who has to change his clothes six or eight times a day on account of drenching perspiration, even if he is at rest. In winter the perspiration is much less. Drugs, such as belladonna, are of no avail. The condition is very troublesome for his everyday life. What is the best treatment for this condition?

REPLY.—In any case of generalized hyperidrosis, the first essential is to exclude the possibility of any disease such as thyrotoxicosis, tuberculosis, and diabetes mellitus. Occasionally, also a septic focus such as chronic sinusitis or infected tonsils may be responsible. When these potential causes have been eliminated, the hyperidrosis is most likely to be due to emotional stress which may require investigation by a psychiatrist or may respond to treatment with a sedative drug such as phenobarbitone or a tranquillizer.

The treatment of such cases of hyperidrosis by internal means is almost invariably disappointing. Many drugs including 'prantal', propantheline bromide ('pro-banthine') and hexamethonium have been used but are of little practical value. Local symptomatic treatment may have some effect, e.g. the application of 6% to 12% solution of aluminium acetate B.P. with cotton-wool, morning and evening.

G. A. GRANT PETERKIN, M.B.E., M.B., F.R.C.P.ED.

Treatment of Shock

QUERY.—It is stated in most textbooks of medicine that the patient with shock should be kept warm in order to combat the shock, but it seems now to be the trend to stop warming shocked patients, on the assumption that more blood is needed in the general circulation instead of wasting it in the skin capillaries by vasodilatation. Do you think it is better to cool the patient so that cutaneous vasoconstriction will cause more blood to return to the heart?

REPLY.—It appears at first sight to be logical to treat by heat the patient whose skin feels cold. The basis for the orthodox treatment of shock by heat, however, depends upon a theoretical approach developed during the 1914-18 War. It was known that 'wound shock' was associated with a low blood volume but it was considered that the blood was dammed up in the capillaries and that the objective of treatment was to get it back into the circulation. Heat was regarded as a stimulus which could mobilize the stagnant blood in the capillaries. To this was sometimes added friction to massage the blood back from the capillary bed.

In fact, the patient who is cold and pale from a shut-down of the capillary blood supplied to the skin is insulating himself against heat loss, and if he is shivering is creating heat. The cold shocked patient may have a raised internal temperature. The low blood volume in 'wound shock' is in fact due to hæmorrhage from the wound or into the tissues or body cavities. Blood transfusion is often the most important part of treatment. It is quite certain that any excessive heating tends to make the patient worse, and there is a considerable body of evidence to suggest that the seriously injured patient is better off cold than artificially heated in any way. When transfusion is carried out at normal room temperature the patient will warm up spontaneously.

Heat is harmful and certainly unnecessary in the treatment of established shock. A blanket may be used to protect a patient from the elements when he is complaining of cold. In addition, hot cradles, hot-water bottles, or radiant-heat lamps can very easily burn the injured patient, particularly if he is not fully conscious. The artificial use of heat interferes with the evidence which can be gleaned from skin colour and temperature. This evidence can be of great help in the assessment of the general response of the circulation and, in particular, of the state of the circulation to an injured limb whose blood supply has been damaged. In the treatment of injured patients heat is harmful, unnecessary, confusing, and sometimes difficult to organize.

There is some evidence to suggest that cooling may play an important part in the treatment of certain severe injuries, and it is fair to say that its value has already been established for certain head injuries.

A RUSCOE CLARKE, M.B.E., M.B., F.R.C.S.

Physical Standards for the Royal Navy

QUERY.—Could you help me with the following problem which has been presented to me by a patient:—

He was born in 1943 and wishes to join the Royal Navy and in order to do so he tells me that he has to sit a naval scholarship at the age of 16½. He is a well built, healthy, intelligent boy in every respect, but he has a pigeon chest and he wonders whether he would be accepted because of this slight deformity. His mother, who is a widow, feels that if he were to work for, and succeed in passing, the scholarship and then be turned down for medical reasons it would cause a great deal of disappointment to the boy, and she would like to know something about his chance of success well beforehand.

REPLY.—The Royal Navy, as indeed is the case with the other Services, has regulations which set forth the defects which may cause rejection of a candidate. In the case of the Royal Navy Scholarship Scheme the following relevant defects may cause rejection: 'A weak constitution, imperfect development, important malformation, or skeletal deformity; and a range of chest expansion below 2 inches'. In every case the decision as to what constitutes an 'important

deformity' depends upon the prognosis as to functional efficiency.

It is, of course, impossible to anticipate the findings of the Admiralty Medical Board, but it would seem likely that, in this case, if the deformity is not gross and there is no limitation of normal function, the boy is likely to be found fit.

Any doctor who would like a copy of the regulations on Physical Standards or who has any questions arising from these standards in relation to individual cases should write to:—

The Medical Director-General of the Navy,
Medical Department of the Navy (Admiralty),
Queen Anne's Mansions, London, S.W.1.

W. D. W. BROOKS, C.B.E., D.M., F.R.C.P.
Consulting Physician to the Royal Navy

PRACTICAL NOTES

Neonatal Respiration

FIVE prerequisites are laid down by Ian Donald (*British Journal of Anæsthesia*, December 1957, **29**, 553) for the satisfactory onset of respiration in the new-born infant: (i) a competent respiratory centre; (ii) a patent airway; (iii) an efficient peripheral circulation; (iv) adequate respiratory movements; (v) the quality of the atmosphere to be breathed. Of the last, he says: 'Honest to goodness air is adequate for most babies and would suffice for all, provided that the other four conditions were met. It is where respiratory activity is insufficient to maintain life that enrichment with oxygen is called for. Nobody now considers that admixture of carbon dioxide serves any purpose as a respiratory stimulant at birth. The asphyxiated child is already poisoned to death with it'. Pure oxygen can be used in the immediate emergency, but should be replaced as soon as possible by concentrations not exceeding 40%, in order to reduce the risk of inducing retrolental fibroplasia in the premature.

On maintaining a patent airway, Donald has this to say: 'The traditional mucus catheter, as used by midwives, is a dirty and inefficient affair and the amounts of material removed by this instrument seem to be a poor quantitative return for all the noisy blowing and sucking that accompany its use. . . . The one obvious benefit from this ritual is the shock of outraged surprise which it inflicts upon an otherwise docile and perhaps too sleepy infant so that it coughs and splutters'. Suction may be lifesaving, but it must be adequate—which means by a mechanical device 'such as a venturi attached to a gas cylinder which is instantly available, or by piped suction from a wall fitting'.

The Ham Test for Occult Blood

As the result of a clinical comparison of the Gregersen, Adler, and Ham tests for occult blood in fæces, H. A. F. Dudley and I. MacLaren (*Scottish Medical Journal*, 1957, **2**, 464) conclude that the Ham test is the most reliable, both in the avoidance of false positives and in the prediction of the correct clinical status of the patient.

Ham's reagent is made by dissolving 1 g. of benzidine dihydrochloride in a mixture of 50 ml. of absolute alcohol, 20 ml. of glacial acetic acid and 30 ml. of distilled water. The test is carried out by mixing 0.25 ml. of this reagent with smears of fæces on filter paper, obtained by digital examination of the rectum. A similar quantity of 10% hydrogen peroxide is then added. In the presence of blood, blue coloration develops within a minute.

In a series of 40 tests on eight orthopædic inpatients, in none of whom was there any evidence of gastro-intestinal disease, 26 positive results were obtained with the Gregersen test, 21 with the Adler test, but only five with the Ham test. In a small series of seven patients thought to have a perforated peptic ulcer, six positive results were obtained with the Ham test. At operation, acute cholecystitis was found in the negative case. In six patients with acute colonic obstruction there were four positive results with the Ham test, and in all four a malignant obstruction was found at operation. The causes of obstruction in the two negative cases were diverticulitis and volvulus of the sigmoid colon.

Treatment of Napkin Rash

SATISFACTORY results are reported by A. Lowry (*Nursing Mirror*, December 13, 1957, **106**, 802) from the use of 'metanium' ointment and powder in the treatment of napkin rash in a series of 25 babies, aged eight weeks to 25 months, in a London day nursery. The composition of the ointment is:—

Titanium tannate	0.1 part
Titanium borate	5 parts
Titanium oxide	12 ,,
Titanium peroxide	5 ,,
Titanium salicylate	3 ,,
in excipient.	

The powder contains the same salts, together with talc and aluminium silicate.

After routine cleansing the ointment was applied over the entire affected area, 'a small amount being sufficient to leave a thin, pinky-white film on the skin'. This film has waterproof properties and provides a good base for the subsequently applied powder. In this way 'the skin is protected from the moisture and the

acids or alkalis of the urine for up to three hours after application', whilst 'the layer of powder enhances the soothing effect of the titanium salts'. Healing was complete within three days in four cases, in ten days in 16 cases, in thirteen days in two cases, and by the 17th day in one more. Only one case did not respond, and in one case there was delayed healing on account of a concurrent diarrhœa.' No toxic effects were encountered, and both ointment and powder are described as 'pleasant and simple to use', and leave no stains.

Treatment of Plantar Warts

DIATHERMY under general anæsthesia is the form of treatment for plantar warts in children recommended by R. B. Coles (*Public Health*, January 1958, **71**, 371). Gas and trilene anæsthesia is used, and the wart is pared down and then destroyed with monopolar diathermy. The procedure is quite a simple one and 'it is quite easy to do six cases in forty minutes'. The patients are asked not to cover the dry desiccated lesion. In a few days the area is quite painless, and in about a fortnight it is possible to pare away the dead wart material, when occasionally the resulting cavity requires a little gentian violet dressing. In some cases a blood blister may develop underneath the wart during the immediate post-removal stage, and this may need incision and evacuation. In a recent series of 156 cases of plantar warts, 22 resolved spontaneously, and the author treated 128 of the remaining 134 cases by this method. Sixty-two of them found the wart comfortable twenty-four hours after treatment, and in the other cases the average duration of pain was 2.6 days. All the lesions were healed within four weeks of treatment. Three patients required a repeat treatment: either because new warts had appeared or because the original one was inadequately destroyed.

Iron-Dextran in Anæmia in Children

'INTRAMUSCULARLY administered iron-dextran complex, when used in the proper dosage in the treatment of *bona-fide* cases of iron-deficiency is a safe and rapidly effective method of correcting such a deficiency', according to W. H. Bartlett and E. C. Beatty, Jr. (*American Journal of Diseases of Children*, December 1957, **94**, 662). 'With this simple and effective method', they contend, 'there should never be need for the potentially hazardous and relatively expensive blood transfusion in the treatment of simple, uncomplicated iron-deficiency anæmia'. The indications for its use in children are: intolerance to, or poor absorption of, orally administered iron; gastro-intestinal disease adversely affected by orally administered iron; the failure of orally administered iron to create adequate iron stores; unreliability of the mother for supervision of oral administration. The authors' conclusions are based upon their experience in a series of 65 infants and children, aged 2 months to 7 years, whose hæmoglobin was below 8.5 g./100 ml. of blood. The lowest level in the series was 4 g./100 ml. Dosage was based upon one of the following formulæ:—

Mg. elemental Fe = B.W. × [16 − (Hb × 1.3)]
or Mg. elemental Fe = B.W. × 1.5 (13 − Hb)
where B.W. = weight in lb. and Hb. = hæmoglobin in g./100 ml. of blood.

Inpatients received consecutive daily doses, and outpatients consecutive weekly doses of iron-dextran complex ('imferon'). This was given by deep intramuscular injection in the upper and outer quadrant of the buttock. A maximum single dose never exceeded 200 mg., and more than 100 mg. was never given per injection site. No side-effects were encountered, and pain at the site of injection was 'comparable to that of a penicillin injection of equal volume'.

Hydrocortisone Snuff in Hay Fever

'INTRANASAL insufflation of hydrocortisone offers another effective, easily administered form of therapy for the patient with seasonal allergic rhinitis not controlled by conventional means', according to C. F. Lake and his colleagues (*Proceedings of the Staff Meetings of the Mayo Clinic*, October 30, 1957, **32**, 641). This conclusion is based upon the findings in 18 patients (15 adults and 3 children) with ragweed hay fever, who had not responded to hyposensitization and/or antihistamine therapy. Adults received a daily dose of 15 mg. of hydrocortisone, two of the children received 5 mg. daily, and the third received 7.5 mg. daily. Each patient was instructed to use the inhaler type of powder dispenser three or four times daily. The results showed that six patients achieved complete relief, eight were improved to the point that they had only minimal residual symptoms, and three obtained 50% relief. Only one failed to respond. The improvement began in three to five days after the use of hydrocortisone powder was started. No symptoms of hypercortisonism were noted, nor did any evidence of nasal infection occur during the course of the treatment

Corticosteroids in Pemphigus

'THE corticosteroids have proved to be the treatment of choice' in pemphigus, according to M. J. Costello, L. Jaimovich, and M. Dannenberg (*Journal of the American Medical Asso-*

ciation, November 9, 1957, **165,** 1249). This conclusion is based upon their experience in the treatment of 52 cases of pemphigus, more than three-quarters of whom were over the age of 50. The mortality in this series was 33%, compared with over 90% in the 270 cases of pemphigus treated in the 40-year period preceding the introduction of steroid therapy. In severe cases the initial treatment was either the daily intravenous administration of 25 units of corticotrophin in 500 to 1000 ml. of 5% dextrose, or the injection of 40 units of repository corticotrophin twice daily. In 'less urgent but moderately extensive eruptions', twenty 5-mg. tablets of prednisone were given daily for a week and the daily dose then gradually reduced. 'The majority of patients', it is said, are finally free of lesions with a maintenance dose of three or four tablets (15 or 20 mg.) daily. It was found 'advantageous' to augment this routine with 40 units of repository corticotrophin intramuscularly once or twice a week. A warning is given that 'it is unwise to try to rid the patient of every last lesion by continuing his treatment on even a moderate dose schedule' because of the risk of complications. Side-effects were common, and there were two deaths from gastro-intestinal hæmorrhage. The best results were obtained in pemphigus vulgaris and pemphigoid.

Controlling Toxicity of Streptomycin

PANTOTHENIC acid, in doses of 50 mg. thrice daily, is of value in controlling the neurotoxic symptoms of streptomycin, according to A. C. Penman and his colleagues (*Tubercle*, December 1957, **38,** 422). Among 31 patients who developed symptoms of vestibular damage during streptomycin therapy, symptoms improved in all but one, following the administration of pantothenic acid, and in 26 they disappeared entirely. Four of the patients who improved were discharged from the sanatorium too soon after the initiation of treatment to be able to decide whether the pantothenic acid would have produced complete relief of symptoms. It is emphasized that pantothenic acid was given at the first sign of trouble and before much vestibular damage could have occurred, and therefore the findings have no direct relevance to the possible value of pantothenic acid in well-established cases. In the authors' experience, neither age, sex, nor duration of streptomycin therapy had any bearing upon the efficiency of pantothenic acid in relieving the symptoms of early vestibular disturbance.

Antibiotics in Dentistry

'IN oral treatment the claimant indications for antibiotics are comparatively few', according to R. Goulding (*British Dental Journal*, December 3, 1957, **103,** 379). Of the various ulcerative conditions of the mouth, only Vincent's infection 'can be depended upon to respond to antibiotic measures, and penicillin is the drug of choice'. For aphthous ulceration, acute herpetic gingivitis, and lichen planus, 'the most that can be hoped for is relief, certainly not cure', from local application or mouth-washes of a broad-spectrum antibiotic such as chlortetracycline. Routine insertion of antibiotics into extraction sockets is frowned upon except possibly after the removal of deeply impacted lower third molars. For root canal therapy, 'the prevailing opinion is that topical antibiotics ensure better results'. Systemic administration of antibiotics is definitely indicated 'prophylactically against bacterial endocarditis when oral surgery is to be performed in patients with congenital and rheumatic heart lesions, for actinomycosis and osteomyelitis, for spreading cellulitis of dental origin with systemic reactions, and for fractures of the jaw'. As the majority of organisms giving rise to oral infections are penicillin sensitive, this is the antibiotic of choice. For Vincent's infection, penicillin chewing-gum is preferred to penicillin lozenges —a piece containing 5000 units to be chewed every three to four hours.

Palmar Pulsation

PALMAR pulsation is a useful confirmatory sign of patent ductus arteriosus in young children, according to D. Stuckey (*Medical Journal of Australia*, November 9, 1957, **ii,** 681). In a series of 22 cases in children, aged 0 to 12 years, it was present in 21. The main value of this sign, however, is in the 0- to 2-year age-group, as it becomes an increasingly common phenomenon in health as age increases. Thus, it was found in only two out of 32 normal children aged 0 to 2 years, but was present in five out of nine normal children aged 8 to 12 years. In a series of 40 children aged 0 to 2 years, with congenital heart disease other than patent ductus arteriosus, it was present in only one case. The pulsation is closely associated with the pulse pressure. Thus, in a series of 86 children, whose pulse pressure was 50 mm. Hg, or less, palmar pulsation was present in only 10, whereas in 20, whose pulse pressure exceeded 50 mm. Hg, it was present in all but one. The pulsation is present in the superficial palmar arch as it crosses the heads of the metacarpals, when it can be easily felt as a strong pulsation. The only other conditions likely to be associated with palmar pulsation in children are fever, severe anæmia, and high environmental temperatures (i.e. exceeding 80° F. [26.7° C.]).

REVIEWS OF BOOKS

The Diagnosis and Treatment of Infections. By D. Geraint James, M.D., M.R.C.P. Oxford: Blackwell Scientific Publications, 1957. Pp. viii and 234. Price 30*s.*

This book is divided into three separate sections. The first describes the chemotherapeutic agents in general use, with special emphasis on the antibiotics. The properties of these substances are described, their fate and modes of action are defined and their toxicity assessed. The second part deals in a similar synoptic way with the commoner micro-organisms which cause human disease and ranges from the metazoa to the viruses. The last section describes infections as they present in the different systems of the body and discusses their diagnosis and treatment. The book concludes with a brief account of the methods of investigating pyrexia of unknown origin and of the use of corticosteroids in the management of infections. The author aims at a synthesis of the disciplines of the clinician and laboratory worker and the work is said to be especially for the benefit of the student and the practitioner.

This rather unusual approach to the diagnosis and treatment of infections has been more successful than might have been anticipated. The author has obviously had some difficulty in avoiding repetition but this has been reduced to a minimum and the book is extraordinarily succinct and at the same time comprehensive and up to date. Although it is not likely to be read consecutively, except by a student in the throes of examination fever, almost every page has some point of interest for the practising clinician. The numerous tables make it a satisfactory book for quick reference and there are few practitioners who would not find it a useful addition to their library.

Cortisone Therapy. By J. H. Glyn, M.D., M.R.C.P., D.Phys.Med. London: William Heinemann Medical Books Ltd., 1957. Pp. x and 162. Figures 4. Price 21*s.*

In this small book Dr. Glyn has undertaken a very difficult task and it is greatly to his credit that he has achieved a remarkable measure of success. The place of cortisone, hydrocortisone and the newer steroid derivatives, prednisone and prednisolone, in the treatment of rheumatoid arthritis and other diseases of connective tissue has been the subject of much controversy during recent years, and the last word has certainly not been spoken. Dr. Glyn reviews developments since Hench's discovery of cortisone in 1949 and then proceeds to describe what is known of the nature and pharmacology of cortisone and other steroid drugs. Practical problems of therapy are discussed in great detail and with the authority of extensive personal experience. A chapter is devoted to the use of cortisone in diseases other than rheumatoid arthritis. The indications for, and the technique of, intra-articular injection are described.

This monograph provides a valuable appraisal of the present position in regard to steroid therapy. It should be welcomed not only by physicians primarily interested in the rheumatic diseases, but by general practitioners whose experience in the use of these powerful hormones has of necessity been more limited, but who are now free to prescribe them.

Burns: Pathology and Therapeutic Applications. By Simon Sevitt, M.D., M.Sc., F.R.C.P.I., D.P.H. London: Butterworth & Co. (Publishers) Ltd., 1957. Pp. xii and 364. Figures 85. Price 36*s.* 6*d.*

The author of this treatise is consultant pathologist to the Birmingham Accident Hospital and the burn research unit of the Medical Research Council in that Hospital and speaks with the authority of almost ten years' intensive study of most of the problems encountered in burns. The book is a laudable attempt to deal comprehensively, within comparatively small compass, with the well-known and also the problematical aspects of the pathology of the burned patient and to point out the therapeutic applications. Following chapters on the physics of heat transfer and the histological changes which take place in burned skin, the usual reaction of burn trauma is described. The author deals in some detail with the hæmodynamic and circulatory effects of burns shock, electrolyte balance and the other miscellaneous factors present in this phase. Morbid anatomical changes are fully described. There is an interesting analysis of mortality and the causes of death, which emphasizes again that improved understanding of shock and its treatment has not resulted in the decrease in mortality that might have been expected. The extensively burned patient now survives shock, often only to die later. The author deals with endocrine responses, in particular that of the adrenal gland, and reviews recent concepts which have originated on this aspect of the problem.

Although he has carried out a comprehensive review of the literature on burns, the subject is now so extensive that it cannot be fully dealt with in a volume of this size and at times clarity has had to be sacrificed to brevity. Nevertheless this book contains much valuable information and collates knowledge which heretofore has not been easily available. It should

prove valuable to postgraduates as an excellent review and guide to further study.

An Introduction to Chest Surgery. By GEOFFREY FLAVELL, M.R.C.P., F.R.C.S.ED. Oxford University Press, 1957. Pp. xiv and 354. Figures 49. Price 30*s.*

THE reason given by Mr. Flavell for writing this book is that, as a student, he found medical books cost too much to buy, and were difficult to read. His book is relatively cheap, and is written in a style which will appeal to some students: it is dogmatic, brief, and in places racy ('. . . the moving ribs are the fingers which play the accordion of the lungs . . . '). It is suitable for students, house officers and those studying for higher surgical qualifications. The text is illustrated by a number of simple line drawings most of which are original. There are also many radiographs which have been excellently reproduced. Some of the latter demonstrate conditions which are unusual, but the common lesions are included. The text not only describes the pathology, diagnosis and treatment of common diseases, but includes brief descriptions of preoperative, operative and postoperative techniques.

It is inevitable that a book of this type will contain a number of statements to which an experienced thoracic surgeon would take exception, but on the whole the author succeeds in his purpose; and within the limits defined the book will add to general medical knowledge.

Fat Consumption and Coronary Disease. By T. L. CLEAVE, M.R.C.P. Bristol: John Wright & Sons Ltd., 1957. Pp. 40. Price 5*s.*

SURGEON CAPTAIN CLEAVE applies to coronary disease the views concerning the inevitable wisdom of natural instincts that he has advanced elsewhere in regard to disease in general. He is not so much concerned in proving the relationship between fat consumption and coronary disease, as with studying the application of the Darwinian principle to this relationship. He holds that the extreme irregularity in the incidence of coronary disease in various countries, which seems to bear no relation to the total fat consumption in those countries, is explained by an examination of what he terms the unnatural fat consumption in those countries. He holds that man may eat with perfect safety any fats existing in his natural environment, so long as he eats them in strict proportion to his appetite. In civilized countries there is, however, through the agency of arbitrary meals and arbitrary food mixtures (of which ice-cream is one) a profound interference with this instinct. Coronary disease is very rare in races living under primitive conditions whether, like the Bantus, they eat minimal amount of fats or, like the Arcti Eskimos, they have a diet as rich in fats as an race in the world.

This small book is the work of an enthusias It gives the reader much to think about.

The Forequarter Amputation. By H. MOSELEY, D.M., M.CH., F.R.C.S. Edin burgh: E. & S. Livingstone Ltd., 195 Pp. viii and 79. Figures 25. Plates 1 Price 42*s.*

THE forequarter amputation is seldom neede today, and the surgeons who have performe or who are likely to perform, it are few. On th other hand the operation, when needed, mu be done early and done well. Mr. Moseley monograph deals with the history of the oper tion, the anatomy of the shoulder girdle, th indications for forequarter amputation toda and the technical details of the operation. Th book will therefore be of real help to a surgeo who contemplates doing the operation for th first time. It is well written, well illustrated, an well produced, and it should be on the shelve of any department of surgery, beside that classi masterpiece, A. K. Henry's 'Extensile Ex posures'.

Current Surgical Management. Edited b JOHN H. MULHOLLAND, M.D., EDWIN H ELLISON, M.D., and STANLEY R. FRIESEN M.D. Philadelphia and London: W. B Saunders Co., 1957. Pp. xxvii and 494 Illustrated. Price 70*s.*

THIS book is an agreeable change from the usua surgical textbook by multiple authors, in whic the editors seek unanimity. They have chosen number of controversial topics, and have picke well-known surgeons to present divergent views The discussions form instructive reading for th experienced surgeon. They would be even bette if the editors, having picked the subjects, ha selected the surgeons for their ability to writ concisely as well as for their eminence.

Mental Deficiency. By L. T. HILLIARD, M.B., D.P.M., and BRIAN H. KIRMAN, M.D., D.P.M. London: J. & A. Churchill Ltd., 1957. Pp. xvi and 517. Figures 90 Price 60*s.*

THIS book is an outstanding contribution to the literature on the subject and seems likely to become the standard textbook of the future fo all serious students not only of mental deficiency in particular, but also of psychiatry in general In parts it is so detailed as to fall into the category of reference book but, over all, it is a book for basic study of the problems presented

by mentally handicapped people, in regard to adequate diagnosis and also to appropriate treatment and rehabilitation. The opening chapters set the tone of the whole book, giving a clear exposition of the needs of mentally handicapped people and especially of the mentally handicapped child against the background of existing legal requirements and educational provisions in the community generally. The fact that the need for new legislation has now been accepted and seems likely to be implemented before long makes it probable that new editions and revisions of the text will be necessary at fairly frequent intervals in the next few years; especially as the book itself will do much to stimulate new thought about the needs of the mentally handicapped in the community and their potential contribution to the community. The section on 'the mentally handicapped individual' recapitulates some of the previous section and in some parts fails to make it clear whether the clinical picture being presented is complicated by the mental handicap of the patient or is a picture of, for example, a physically handicapped child or a mentally disturbed child whose intellectual endowment is at least average. It would be unfortunate if psychosis in childhood were to be studied only in the context of this book, and in fact the chapter on 'the mentally disturbed child' is written on so superficial a level as to be out of place in a textbook where the main topic is dealt with so exhaustively and with such attention to detail.

Despite these criticisms, this book is undoubtedly first class and its publication is a landmark in the history of the care and understanding of the mentally handicapped. It should become standard reading for all medical students and postgraduates whose work brings them into contact in any capacity with mental deficiency.

The Clinical Examination of Patients with Organic Cerebral Disease. By R. KLEIN, M.D., and W. MAYER-GROSS, M.D., F.R.C.P. London: Cassell & Co. Ltd., 1957. Pp. xiii and 96. Price 15*s*.

TESTS of the state of functioning of the higher levels of neuronal integration are not as easy to devise, apply or interpret as the familiar tests concerned chiefly with the sensori-motor levels. Perhaps this is one of the reasons why the more subtle organic disturbances of mental functions may sometimes be overlooked in the course of routine clinical examination. Advances in knowledge suggest that the marginal clinical territory common to neurology and psychiatry is more extensive than it was formerly thought to be. There is therefore a need for presenting in one volume, designed for the use of the practical clinician, a summary statement of useful tests of higher neuronal functions. This book meets this need admirably. Starting with the already familiar tests of disturbance of speech and language function, the authors take us on through disorders in the visual sphere, including disturbances in spatial orientation; disorders of the time sense; dyspraxias and disturbances of special manual skills; disorders connected with subjective disturbances of the body scheme and rules of dominance (laterality). They conclude with a short account of tests of attention, memory and the state of consciousness as a whole.

The book is concise and clear. Practice predominates over theory, although the lines on which interpretations of practical findings may be sought are clearly indicated. It will form a valuable addition to the armamentarium of those whose clinical work lies in the neuro-psychiatric field, and it will be helpful also to the general practitioner and senior medical student.

Essentials of Chemical Pathology. By D. N. BARON, M.D. London: English Universities Press Ltd., 1957. Pp. xi and 247. Figures 28. Price 25*s*.

SINCE the establishment of the National Health Service, laboratory facilities available for the practitioner have multiplied to a remarkable degree, and with this development there have been particularly rapid advances in chemical pathology. Whilst a few elementary student's textbooks on this subject have already been published, and a rather larger number of erudite but weighty tomes that few practitioners have time to read, Dr. Baron's little book is the first to provide a concise and reasonably full account of recent progress and accepted knowledge in this field. The first five chapters deal with the normal and abnormal biochemistry of the body fluids, electrolytes, carbohydrates, proteins and lipids, whilst the succeeding six chapters are concerned with the chemical pathology of special systems and organs.

It is not a book of laboratory technical methods although some chemical procedures are described in detail to enable the practitioner to make adequate arrangements for the preparation of his patients and the necessary collection of specimens. Methods for ward urine testing as well as tables of normal analytical values are provided. The book is remarkably complete and will be invaluable not only to the specialist in the subject but also as a reference book for all practitioners, whether in consultant or general practice. The author and his publishers are to be congratulated on providing this useful little book at so reasonable a price.

NEW EDITIONS

Eden & Holland's Manual of Obstetrics, by Alan Brews, M.D., M.S., M.R.C.P., F.R.C.S., F.R.C.O.G., in its eleventh edition (J. & A. Churchill Ltd., 63*s*.), enters upon its second half-century with the reputation of being one of the best practical guides to the art and science of obstetrics. As it is only four years since the last edition, little radical change has been required, but many new illustrations have been added. A useful addition is an appendix on emergency obstetric units.

A Handbook on Diseases of Children, by Bruce Williamson, M.D., F.R.C.P., in its eighth edition (E. & S. Livingstone Ltd., 27*s*. 6*d*.), remains very readable and practical, and particularly valuable in those sections (such as the chapters on the heart) in which the author is obviously specially interested. The book first appeared nearly a quarter of a century ago and has maintained a popular appeal. The two chapters on the common fevers are a clever summary, and the various sections at the end of the volume, on infant feeding, infant welfare and drugs, make for easy reference.

The Early Diagnosis of the Acute Abdomen, by Sir Zachary Cope, M.D., M.S., F.R.C.S. (Oxford University Press, 18*s*.).—The eleventh edition of this well-known textbook in thirty-five years is more than a re-issue. The clear advice and lively style, and the frequent and helpful diagrams that have made it so popular, remain, but there are new chapters on the general principles of diagnosis and on vascular lesions in the abdomen. Many of the chapters have been rewritten in part, and some first-class skiagrams have been added. This is a really useful book for students, surgeons, and even more for practitioners.

Oral Medicine, by Lester W. Burket, A.B., D.D.S., M.D. SC.D., in its third edition (Pitman Medical Publishing Co. Ltd., £5), has become a standard work. Because of the development of new methods of treatment and increased knowledge, it has been found necessary to rewrite many chapters and revise others. Moreover, the bibliography also has been completely revised. This work covers not only what in this country would be termed 'medicine for dental students', but also much of 'surgery for dental students' and also in considerable detail oral diagnosis and oral pathology. Its scope is therefore wider than at first sight might be expected, but this is a decided advantage in bringing oral medicine into its true perspective. It is not likely to be of great value to the general medical practitioner, though he will find its perusal interesting. It is a book primarily for the dental specialist, who will find it not only of great interest, but also a most useful and constant source of reference.

Lumbar Puncture and Spinal Analgesia, by Sir Robert Macintosh, D.M., F.R.C.S., F.F.A.R.C.S., second edition (E. & S. Livingstone Ltd., 25*s*.).—The new edition of this admirable little book is excellent, and the author states in the preface that the script has been improved and that some of the illustrations have been made more accurate. The text is profusely illustrated and colour has been used generously to good effect. There are one or two minor points upon which comment can be made, but they are not really such as to detract from the general excellence of the book. The author refers to 'xylocaine' and 'nupercaine', but perhaps in a book of this nature it would be better to give the official names, lignocaine and cinchocaine. It is also stated that cinchocaine is mixed with 5% glucose, whereas the actual figure is 6%. The book is well produced, and should be in the hands of every anæsthetist and physician who is interested in lumbar puncture.

Preventive Medicine and Public Health, by Fred Grundy, M.D., M.R.C.P., D.P.H., third edition (H. K. Lewis & Co. Ltd., 25*s*.).—It is difficult to speak otherwise than well of this book which is to all intents and purposes alone in its field and which achieves its author's purpose very successfully. Professor Grundy writes clearly and succinctly and succeeds in making plain that incredibly chaotic and irrational thing, the local and central governmental machinery of the health services—no mean feat. About one-third of the book is given to a description of the health services and their management, another third to statistics and the control of infectious disease, and the remainder to discussion of principles and to historical considerations. There are some omissions and compressions in the descriptive sections which might be remedied—there is, for example, no description of school medical inspection and only the barest mention of services for handicapped pupils. The section on statistics is clearly a part of the book where the author is very much at home and is an excellent practical introduction to its subject. We forgive its only obvious flaw, over-reliance on the traditional five social classes of the Registrar General's returns, but would suggest that the next edition of the book might mention that this classification, though at present the only one available, has lost a good deal of its validity in recent years.

The contents of the March issue, which will contain a symposium on 'Domiciliary Care', will be found on page A124 at the end of the advertisement section.

Notes and Preparations see page 267.
Fifty Years Ago see page 271.
Motoring Notes see page A87.
Travel Notes see page A91.

NOTES AND PREPARATIONS

NEW PREPARATIONS

'ARCOFAC' emulsion provides 'a palatable means of correcting elevated blood cholesterol levels with comparatively small dosage', and may be used prophylactically as well as therapeutically. Each tablespoonful (15 ml.) contains 6.8 g. of linoleic acid, 0.6 mg. of vitamin B_6, and 11.5 mg. of mixed tocopherols (vitamin E). Issued in bottles containing 12 fluid ounces. (Armour Laboratories, Hampden Park, Eastbourne, Sussex.)

'BISMUTH SUPPOSITORIES (GABAIL)' are intended for the treatment of tonsillitis, Vincent's angina, and acute non-specific infections of the upper respiratory tract. It is stated that 'marked symptomatic relief' has been observed within an hour of administration. Each suppository contains 65 mg. of bismuth and sodium tartrate in a bland base. An additional strength (16 mg.) is also available for use in children under 8 years of age. Issued in boxes of six suppositories. (Distributed by the Anglo-French Drug Co. Ltd., 11-12 Guilford Street, London, W.C.1.)

'PLAQUENIL' tablets each contain 400 mg. of hydroxychloroquine sulphate and are intended for the treatment of 'lupus erythematosus, polymorphic light eruption, rheumatoid arthritis, malaria and giardiasis', and are said to be better tolerated than chloroquine or mepacrine. They are contraindicated in the presence of hepatic disease. Issued in bottles of 100 and 500 scored tablets. (Bayer Products Ltd., Neville House, Eden Street, Kingston-on-Thames, Surrey.)

'SEDUMAX' tablets each contain 120 mg. of carbromal, 120 mg. of bromvaletone, 180 mg. of phenacetin, and 2 mg. of aneurine hydrochloride. They are intended for the treatment of premenstrual tension and mild anxiety states, and for insomnia due to neuralgic pain. Issued in packs of 50 and 300 tablets. (Calmic Ltd., Crewe, Cheshire.)

PHARMACEUTICAL NOTES

ARMOUR LABORATORIES announce the introduction of lyophilized 'chymar' brand of chymotrypsin for intramuscular injection, to supersede their preparation of 'chymar' in sesame oil. Available in packs of 3 vials, each containing 5000 Armour units of chymotrypsin, together with 3 x 1-ml. ampoules of diluent. (Hampden Park, Eastbourne, Sussex.)

THE DISTILLERS CO. (BIOCHEMICALS) LTD. announce the introduction of '"distaquaine" V-K tablets', which are 'a chemically improved form of '"distaquaine" V'. Available in bottles of 100 scored tablets each containing the equivalent of 125 mg. of penicillin V acid (as the potassium salt). (Broadway House, The Broadway, Wimbledon, London, S.W.19.)

FILM NEWS

Ergot—The Story of a Parasitic Fungus (16 mm., colour, sound; running time 27 minutes) 'dramatically illustrates the story of ergot as a fungus and medicinal substance', and was made with the cooperation of Sir Henry Dale, O.M., and Professor Chassar Moir. Available on free loan upon application to The Wellcome Foundation Ltd., The Wellcome Building, Euston Road, London, N.W.1.

Topical Application of Antibiotics (Polybactrin) in Neuro-Surgical Practice (16 mm., colour, sound; running time 20 minutes) deals with the application of 'polybactrin' spray in neurosurgery, and was made at the Royal Infirmary, Edinburgh. Further details may be obtained from Calmic Ltd., Crewe, Cheshire.

ROYAL SOCIETY OF MEDICINE

THE next meeting of the Section of General Practice will be held at 8.15 p.m. on Wednesday, February 19, when the subject for discussion will be 'Emergencies in General Practice'. The opening speakers will be Dr. R. M. McGregor and Dr. J. G. Ollerenshaw.

COURSES AND CONFERENCES

Chronic Rheumatic Diseases will be the subject of a 'concentrated week-end course', with lectures, ward rounds and practical demonstrations, to be held on Saturday and Sunday, March 15 and 16, 1958, at the Rheumatism Unit of St. Stephen's Hospital, Fulham Road, Chelsea, London, S.W.10. Full details may be obtained from the Secretary, Fellowship of Postgraduate Medicine, 60 Portland Place, London, W.1.

The International Federation of Gynæcology and Obstetrics will hold its second world congress at the Queen Elizabeth Hotel, Montreal, Canada, from June 22 to 28, 1958. Full details may be obtained from the Montreal Committee, Second World Congress, International Federation of Gynæcology and Obstetrics, 1414 Drummond Street, Suite 220, Montreal 25, Quebec, Canada.

MARLBOROUGH NIGHT HOSPITAL

UNDER the ægis of the National Health Service, a Night Hospital has now been opened, in

association with the Marlborough Day Hospital, for the short-term treatment of cases of neurosis. Both men and women will be accepted. Full details can be obtained from The Medical Director, Marlborough Day Hospital, 38 Marlborough Place, St. John's Wood, London, N.W.8.

STRANRAER HEALTH CENTRE

THIS Health Centre, the first of its kind in Scotland to serve the whole population of a fair-sized town and large surrounding agricultural area, has now completed its second year of existence. Nine general practitioners—seven principals and two assistants—consult in the five suites in the Centre, which is adjacent to the local hospital. The dressings room is staffed by a nursing sister attached to the staff of the hospital, and provides coverage from 8.30 a.m. to 5 p.m. After hours and on Sundays these services are provided in the hospital. All dispensing is done by the seven local pharmacists. The doctors using the Centre have full access to the hospital x-ray department, which is an 'open' one. A child welfare clinic is held weekly. The office staff consists of a secretary and two shorthand-typist/receptionists.

N.C.B. MEDICAL SERVICE

THE National Coal Board is now spending on medical services the equivalent of £2 14*s.* a year for every man employed. The total capital cost of the medical service since vesting date has been £3 million. When the scheme to give periodic chest x-rays to all men and management in and about collieries starts towards the end of this year, the cost per head of the medical service will increase to £2 18*s.* Virtually all entrants to the industry are now examined by the Board's doctors. The number of statutory examinations conducted exclusively by N.C.B. medical officers in 1956 was 27,763. Of these, 3.1% were rejected. In the same year there were more than 5½ million attendances for treatment at the medical centres of the Board. At the end of 1956 the Board had a staff of 77 medical officers and 330 nurses.

LONDON AIRPORT AMBULANCE SERVICE

THE Ministry of Health has issued a statement emphasizing that requests for ambulance transport for patients arriving by air at London Airport are accepted by the Middlesex ambulance control only from the airport medical officer. Any such patient is dealt with in the first instance by the airport medical officer who then makes the necessary arrangements with the Middlesex ambulance control. If treatment or rest is necessary before the patient continues his journey, the patient is normally taken to a nearby hospital where he remains until he is fit to continue his journey. When long-distance rail journeys are involved, the Middlesex ambulance service provides transport between the airport and the railway station, makes the necessary arrangements with the railway authorities and notifies the appropriate local authority so that the patient is met at the station of destination.

TELEVISION AND THE PATHOLOGIST

COLOUR television has been installed at the Walter Reed Army Medical Center in Washington, D.C., as a means of facilitating reports on material removed at operation. The material is sent from the operating theatre by pneumatic tube to the pathology laboratory, where a section is immediately placed in the microscope. A television camera transmits the picture of the section to the operating theatre, where it is viewed by the surgeon who, by means of an audio connexion, is able to discuss the problem with the pathologist in his laboratory.

ASIAN INFLUENZA

LAST summer Professor J. Mulder (*Lancet*, 1957, **ii,** 334) reported the finding of antibodies to the present Asian strain in people aged 80 to 90, in the Netherlands, which suggested that the pandemic of 1957 might be virologically related to that of 1889-90. Confirmation of this now comes from Italy where, in Florence, R. Davoli and O. Bartolomei Corsi (*Sperimentale*, 1957, **107,** 358) found that out of 78 subjects aged over 65, ten (12.8%) were found to inhibit at a low titre ($\leq 1/64$) the hæmaglutination by the strain A-Singapore 1/57, compared with only one out of 196 subjects aged 5 to 50. These observations were made before the epidemic of influenza had broken out in Florence.

RESEARCH IN INDIA

THE report of the Bengal Immunity Research Institute, Calcutta, for the quinquennium, 1951-55, is one of continuing progress. In addition to routine work on the standardization and analysis of raw materials and pharmaceutical products, an extensive research programme has been carried out, including the nutritive value of foodstuffs, the action of antimalarials on enzyme systems, and chemotherapy.

SHAMPOO FOR PEDICULOSIS

SATISFACTORY results from the use of a shampoo of gamma benzene hexachloride ('lorexane no. 3') in the treatment of pediculosis capitis are reported by Dr. J. L. Burn, medical officer of health for Salford, in his annual report for 1956. Sufficient shampoo for two weekly treatments for the whole family, with printed instructions,

and a letter for their parents were given to the infested children at the first examination. Apart from being effective, it was also popular. Both parents and children liked it. 'Parental opinion', according to Dr. Burn, 'was succinctly expressed by one small boy in conversation with a health visitor—"me mam ses it's good stuff—it doesn't 'arf shift 'em" '.

HERBAL SMOKE FOR NEURODERMATITIS

As part of a two-year programme to 'evaluate the traditional Chinese herbal treatment in dermatology', Hu Ch'uan-K'uei *et al.* (*Chin. med. J.*, 1957, **75**, 832) have investigated the value of the following 'herbal smoke' in the treatment of neurodermatitis:—

Dictammus albus	10
Gynocardia odorata	10
Galla sinensis	5
Carpesium abrotanoides	4
Pinus sinensis	4
Sophora flavescens	3
Silver divaricatum	3
Phellodendron amurense	3
Actractylis	3

The powdered compound is placed on glowing charcoal, about 7.5 g. every five minutes, and the smoke is brought to the skin lesions through appropriate flues. Alternatively, a 'herbal powder roll' is used, which may be lighted to generate smoke as well as heat and held close to the lesions. It is claimed that the 'cure rate' with smoke treatment was 82.5%, compared with 89% with x-rays, and 40 to 50% with 'novocain block'.

HEALTH PROPAGANDA IN SINGAPORE

It must have been with mixed feelings that the Singapore health authorities found that police had to be called in to deal with a crowd of what are described as '5000 angry mothers and screaming children' who tried to break into a diphtheria immunization clinic there. There has been an outbreak of diphtheria in the colony recently, and apparently the rush for inoculations was so great that the clinic had to close after an hour. It certainly looks as if the anti-diphtheria propaganda has been successful.

WINE FOR CHICKENS

At a West Germany research institute 60 chickens are being given a daily dose of a third of a pint (200 ml.) of various wines (equivalent to a human consumption of 9 to 10 pints [5 to 5.5 litres]), in order to try and discover whether cirrhosis of the liver is caused by all types of wine. Apparently it has been suggested that wines from hybrid vines—i.e. vines grafted with the North American wild grapevines to increase resistance to vine diseases—may affect the central nervous system and the liver more than wine from other vines.

BAVARIAN SPAS

A booklet has recently been published, giving full details of the large range of spas in Bavaria. It is entitled 'Cures in Bavarian Health Resorts', and copies can be obtained on application to: Bayerischer Heilbäder-Verband e.V., Bad-Kissingen, Germany.

PUBLICATIONS

The Story of Peptic Ulcer, by Richard D. Tonkin, M.D., F.R.C.P.—Whether you have a peptic ulcer or not (and most doctors have one) you will love this book. It is amusingly written, delightfully illustrated, and every word of it is sound common sense. If you have no ulcer you will learn how to avoid one; if you have one you will learn how to cure it. In any case you will want to present a copy to your dyspeptic friends and patients. (W. B. Saunders, price 16*s.*)

The Shoulder, by James Cyriax M.D., M.R.C.P.—Shoulder conditions are not always easy to diagnose and sometimes are notoriously difficult to treat. Dr. Cyriax has attempted to simplify accurate diagnosis by careful attention to applied anatomy and by separate testing of each anatomical structure in the region. Perhaps he overstates his case when he says that 'the consultant with all the facilities of the large hospital is no better placed in reaching a conclusion than the family doctor himself' and that a radiograph is unnecessary. There are few clinicians with experience in the treatment of the stiff and painful shoulder who would agree that shoulder trouble is 'easy to put right' or that its treatment 'is a pleasure'. Access to a physiotherapy department whose members are trained in the methods of Dr. Cyriax is necessary for much of the treatment advocated, and of course this may not be possible, especially in remote districts. (Cassell & Co. Ltd., price 5*s.*)

Rudolf Steiner Education, by L. Francis Edmunds.—Rudolf Steiner education deals not only with handicapped children. In Britain there are seven schools, two of which are formally recognized by the Ministry of Education for children up to university entrance standard. For nearly all children in this country, factual education is remorselessly stamped on the mind by decree. Rudolf Steiner, who died in 1925, provided an alternative, based on the spiritual development of the child; it is co-educational and relieved of 11-plus and similar strains until the final years. This little book describes briefly the purposes and methods of Rudolf Steiner education. It is well written and worth reading by all who wish to know something of this educational experiment. The smaller a method is in proportion to orthodoxy,

the more likely it is to be regarded as a system for cranks. It is hoped that such a fate will not overtake the good features of Rudolf Steiner education; but it will have to prove itself by results, both in academic and other fields. (New Knowledge Books, price 3*s*. 6*d*.)

Sidelights on the History of Medicine, edited by Sir Zachary Cope, is an unimpressive and unrepresentative selection of papers read before the section of the history of medicine of the Royal Society of Medicine during the past forty years, and now published in book form as part of the jubilee celebrations of the Society. A book such as this has no appeal to the expert, and priority should therefore have been given to those papers which would appeal to the general reader. Excellent examples of these, which are included, are Dr. Guthrie's 'The patient: a neglected factor in the history of medicine', Sir D'Arcy Power's 'The fees of our predecessors', and Dr. Lyle Cummins' scholarly essay on Auenbrugger and Lænnec. (Butterworth, price 45*s*. [35*s*. to Fellows of the Society].)

Charles Turner Thackrah, by A. Meiklejohn, is a reprint of Thackrah's classical book on 'The effects of arts, trades and professions . . . on health and longevity', a pioneer work on the subject which was first published in 1832. There is a preliminary account of the life of the author, who was one of the founders of the Leeds Medical School but, in spite of Dr. Meiklejohn's enthusiasm, anything but a likeable character. (E. & S. Livingstone, price 25*s*.)

Medical Writing, by Morris Fishbein, M.D., in its third edition remains one of the best guides on the subject for the doctor who proposes to put pen to paper. It is unfortunate, however, that in his list of abbreviations of names of periodicals the author has not adhered to those recommended in 'World Medical Periodicals', published by the World Medical Association, which is now generally accepted by all the major medical journals of the world. It is also surprising to find the old-fashioned, and now discarded, symbol of the drachm still being used. These, however, are minor criticisms of a book by one with long experience in the sphere of medical editing. If its advice were followed the lot of the medical editor would be much easier. (McGraw-Hill Publishing Co., price 52*s*. 6*d*.)

The Physician's Own Library, by Mary L. Marshall, librarian to the Rudolph Matas Medical Library, Tulane University, will be of interest to medical societies with small libraries, as well as to individual practitioners. It includes a chapter on writing a medical article and one on preparing an index. The only criticism of the book is that it recommends the reprehensible habit of using printed 'mailing cards' for soliciting reprints of articles. (Blackwell Scientific Publications, 22*s*. 6*d*.)

Medical Books, January 1958, gives details of all new and forthcoming medical publications of E. & S. Livingstone Ltd., from whom copies may be obtained upon application to 15-17 Teviot Place, Edinburgh 1.

OFFICIAL PUBLICATIONS

Report of the Ministry of Health for the year ended 31st December, 1956. Part II. On the State of the Public Health.—In this, his annual report for 1956, the chief medical officer of the Ministry of Health notes that deaths from tuberculosis have undergone another reduction by over 1000, as compared with 1955 (5,375 and 6,492, respectively). Deaths from cancer and diseases of the vascular system, however, continue to increase. An interesting feature of the mortality from cancer of the lung is that rates of increase in the two sexes are beginning slowly to approximate. The number of deaths from diphtheria was only eight—the lowest ever recorded in this country. Another new low level was achieved in maternal mortality—the number of deaths of women attributed to pregnancy and childbirth fell to 327. Toxæmia has now assumed first place in importance as a cause of maternal death.

There was a slight increase in the number of new cases of early infectious syphilis attending clinics (884, compared with 737 in 1955), and a comparable rise in the number of new patients in the late and latent stage of the disease (3,707, compared with 3,502 in 1955). The rise in gonorrhœa was more marked—from 17,845 in 1955 to 20,388. 'The increased use of the so-called "tranquillizers" ' is described as 'the most striking though not necessarily the most important development in drug treatment in 1956'. In the opinion of the chief medical officer, 'the so-called tranquillizing drugs are beginning to reveal themselves as double-edged weapons. Misuse of certain of these may cause complications which may have a fatal issue and with regard to all of them the possibility of addiction is always in the background'. (H.M. Stationery Office, price 9*s*.)

Corrigenda. — 'PRIMOTESTON - DEPOT'. — This preparation is manufactured by Schering A.G. Berlin, and distributed throughout the United Kingdom by Pharmethicals (London), Ltd. (See *The Practitioner*, 1958, **180**, 18, 19)

'PROCTOSEDYL' ointment is supplied in 5-g. tubes (*not* 5-mg. tubes, as stated in our last issue, p. 149).

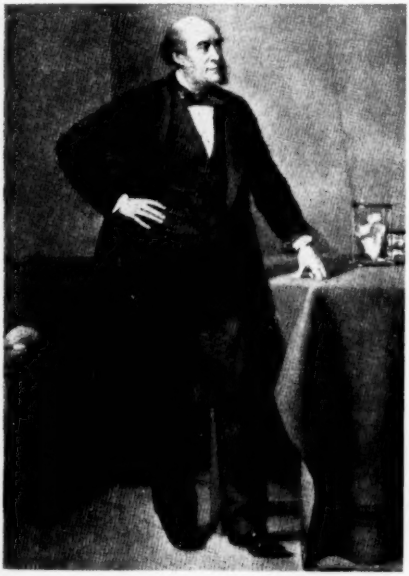
THE PRACTITIONER

Fifty Years Ago

'In the history of the profession there are grounds for the statement that isolation promotes originality'.—William Osler: 'The Army Surgeon' (1894).

FEBRUARY, 1908

THE Nobel Prize for Medicine has been awarded, this year, to a French physician', and 'Notes by the Way' congratulate Dr. Laveran on his 'attainment of an honour so very far from barren. His career, like that of Major Ronald Ross who received the prize in 1902, shows to what excellent scientific use the special experience of an army surgeon may be turned;



Sir William Fergusson, F.R.C.S., F.R.C.S.Ed. (1808-1877)

and it is not necessary to explain that he was Major Ross's principal predecessor in the effective study of malaria. His opportunity came in 1878 when he was stationed in Algeria; and there, after patient and laborious research, he established the fact that the disease was not due, as had previously been supposed, to miasma, but to the presence in the blood of minute protozoan parasites. . . . Subsequently, as if he aspired to proceed alphabetically through the long list of human ailments, Dr. Laveran discovered a microbe believed to be responsible for mumps, and he is now working at the Pasteur Institute'.

The son of a military surgeon, Charles Louis Alphonse Laveran (1845-1922) graduated M.D. at Strasburg in 1867 and in 1878 went with the French army to Algeria where he remained until 1883. In the following year he was appointed professor of military hygiene and clinical medicine at the school of Val-de-Grâce. He joined the Pasteur Institute in 1897.

'There are some branches of medical science that they take more seriously in France than in England. One does not easily picture an English Government commissioning an alienist to attend Revival Meetings in Wales in order to study and report upon "the effect of mysticism upon mental maladies". The French Government did so, and the result is a most interesting and vivacious book by Dr. J. Rogues de Fursac entitled "Un mouvement mystique contemporain". On the whole the author seems to have found the Revivalists rather less mad than he expected, and comparatively little insanity traceable to their hysterical manifestations. . . . A branch of the subject on which Dr. Rogues de Fursac failed to satisfy a very natural curiosity was the effect of the revival on the illegitimate birth rate. We do not ourselves know whether the figures are such as to warrant any inference; but risks of the character indicated are invariably present whenever young men and maidens are touched by the contagion of violent religious excitement'.

The February number of *The Practitioner* fifty years ago opens with 'A Clinical Lecture on the Causes and Operative Treatment of Umbilical and Ventral Hernia' by Arthur E. Barker, F.R.C.S., Professor of Surgery at University College Hospital Medical School. Guthrie Rankin, M.D., F.R.C.P.Ed., Physician to the Seamen's Hospital, Greenwich, writes on 'Infantile Paralysis': 'In the large majority of cases, the disease is established, and the damage done before advice is sought, but, even were it otherwise, nothing in the way of prophylaxis is possible in the present state of our knowledge'.

J. W. Thomson Walker, F.R.C.S., Assistant Surgeon to St. Peter's Hospital for Stone, describes 'The Radical Treatment of Cancer of the Prostate'. W. M. Robson, M.D., M.R.C.P., Assistant Physician to the Northampton General Hospital, discusses 'The Contracted White Kidney', 'because I believe that the medical profession, taken as a whole, does not realise the frequency, or the fatality of the disease'.

R. Tanner Hewlett, M.D., F.R.C.P., Professor of General Pathology and Bacteriology in King's College, London, and H. Batty Shaw, M.D., F.R.C.P., Physician to University College Hospital, review tropical diseases and diseases of the blood respectively.

F. Gardiner, M.D., F.R.C.S.Ed., Assistant Physician, Skin Department, Royal Infirmary, Edinburgh, in his article 'The Treatment of Chilblains', writes: 'Quackery is a plant which, like other weeds, grows readily on neglected soil, and one, therefore, does not wonder at the large number of nostrums which are said to cure this trouble. I am of opinion that no case need go beyond the erythematous stage, that recurrences can be aborted, and that many cases can be cured'. He describes ichthyol and formalin as standing out 'pre-eminently as of value in the early stages'.

The 'Leader in Modern Medicine' this month is Sir William Fergusson, whose 'reputation rests not merely on the fact that he was a brilliant operator, but also on the fact that his system of operative surgery was eminently "conservative". . . . Out of the operating theatre, Fergusson did not shine as a lecturer. This was largely due to his reticence, and imperfect command of abstract subjects, and his views on pathology were somewhat archaic. . . . He was a good violinist, and a devoted disciple of Isaak Walton. Tall, dignified, and of good presence, he was a genial companion and an excellent host'.

'Notes from Foreign Journals' describe 'A New Treatment for Tuberculosis' introduced by Lemoine and Gérard of Lille, 'based upon the antitoxic action of the liver': 'Tuberculous laryngitis has been almost invariably benefited in a short time by intra-laryngeal injections of paratoxine, as the Professors call their product. Diarrhœa, even in the hectic stage, is greatly improved when paratoxine is given by the mouth'.

Among the books reviewed are E. Klein's 'The Bacteriology and Etiology of Oriental Plague' ('profusely illustrated with a number of beautiful plates, but lacks an index'), and F. J. Poynton's 'Heart Disease and Thoracic Aneurysm' ('well written and will take its place as one of the best textbooks on the study of disease of the heart').

'Practical Notes' include an item 'Cardiac Failure in Pneumonia': 'In acute pneumonia, the second cardiac sound over the pulmonary artery is frequently found to be accentuated. This sign is a valuable one, and gives the practitioner an indication as to the condition of the pulmonary circulation. The pulmonary second sound becomes very much less distinct when the right auricle and ventricle become distended, and the right ventricle is unable to completely empty itself. . . . Occasionally, as early as the third day in a case of acute lobar pneumonia, there may be a sudden and early collapse of the heart'.

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